Dietary Guidelines Advisory Committee Meeting

Day 2

Date: October 31, 2008
Time: 8:35 a.m.
Location: USDA South Building
Jefferson Auditorium
1400 Independence Avenue, SW
Washington, D.C.

Meeting Conducted By: Dr. Van Horn
DR. VAN HORN: Good morning. We are going to get started again this morning. Thank you all for joining us and Happy Halloween. We actually had one of our members come in here looking a little more like Halloween, but we’ll let you guess who that is. We are delighted to have a chance to launch into our second round of discussion, but first, for the purposes of those in the audience, we thought it would be helpful to summarize some of the discussion that took place yesterday. Those on the Committee have a copy of these, but I’ll just briefly give you a hint on exactly what the key topics were that were addressed. Some of the major points related to the discussion on nutrient adequacy included the shortfall nutrients for adults and children that continue to be a problem, including calcium, potassium, fiber magnesium and vitamin E; and also for adults, vitamins A and C; and children, especially vitamins A, C and possibly phosphorous were a concern. Shortfalls in terms of food components include fruits and vegetables, especially dark green and orange
vegetables and legumes, whole grains and milk. There is excessive intake of sodium and calories from solid fats, alcohol and added sugars, otherwise known as SOFAAS. I guess we are all calling them that.

Then there is the need to emphasize meeting nutrient needs within energy needs by recommending nutrient-dense foods, and perhaps considering new ways to group foods, and that’s something that we touched on yesterday that we’ll perhaps take up again. I would also add to that actually the whole concept of discretionary calories, which is the flip side of that; the meeting the nutrient density needs, but also considering what discretionary calories might include.

Since diet is a complex exposure, there is a need to emphasize dietary patterns. We talked about whole foods and the cultural and social aspects of eating and the needs for including dietary patterns, even along with healthy aging and longevity.

Then we raised some potential new questions; looking at the amount and source of protein; the role of bioactive proteins in methyl groups; in the discussion of folate, we talked about potential dual
effects on cancer and risk, and needs to look at the
positives and negatives of that; vitamin D, of course,
which is currently a hot topic and the need to consider
it, and to look in combination and coordination with
the IOM Committee. Other nutrients that were not
investigated in 2005 include things like selenium and
possibly other micronutrients. And then, the whole
concept of bio availability that we raised. And then
also, the need to look at changes in nutrient content
over time in processing and the whole concept of
globalization of food and nutrient integrity; so, the
idea of looking at possible speakers to give us further
input on that.

Then, in our fluid and electrolyte discussion, we
talked about the recent research that continues to
recognize the benefits of potassium and reduced sodium
in the diet. Consensus still exists on the benefits of
lower blood pressure and the fact that blood pressure
rises throughout one’s lifetime, which is usual, but
not normal in that ideal. The adverse effects of
excess salt intake, of course, include hypertension and
cardiovascular disease and stroke risks; probable
relationships with gastric cancer; and there is a relationship -- a suggested relationship of increased risk of osteoporosis and increased left ventricular mass with higher sodium intakes; and then the hypothesized relationship with overweight and obesity as well, recognizing that more food obviously contributes more sodium.

So, looking at new evidence and emphases, blood pressure status of Americans is getting worse. We assume its related to the obesity epidemic; evidence and benefit of reducing salt and increasing potassium, as far as cardiovascular disease events; and concerns about blood pressure in children, which continue to accompany the rise in pediatric obesity as well. Some potential new research questions; what dietary factors influence blood pressure in children and young adults; not necessarily assuming that everything that’s true in adults is true in children; we need to take a closer look at their diet. Should the target for sodium intake be reduced from 2,300 to 1,500 milligrams per day, or at least in those at high risk or already hypertensive or pre-hypertensive? Looking at the
current sources of sodium; the effects of certain beverages, such as coffee and tea on cardiovascular disease and its risk factors; what are the effects of sugar-sweetened beverages and artificial-sweeteners, and water on weight in children and adults. So, the discussion then revolved around concern about blood pressure in children, differentiating inherited blood pressure hypertension versus acquired through environmental exposures, and looking at again, high sodium/low potassium intake. More discussion on coffee, tea and considering other beverage-related questions, as time goes on, related to blood pressure. Also, the effects of reduced sodium on protein balance, iodine deficiency and food safety questions related to making sure we don’t compromise any of that. For potassium, does it make a difference, if potassium comes naturally or is added as a supplement? We spent a little time on that. And then additional information may be needed on the composition of processed foods and new fortified products; has food composition data been updated with these new foods, such as chicken injected with brine
and some of the other processed foods that we kind of
take for granted?

So, having summarized all that, I guess we had a
pretty productive day yesterday, and I’m sure today
will be equally productive. I am happy now to welcome
Dr. Pi-Sunyer, as we start off this day’s discussion
related to energy balance and weight maintenance,
management. Oh, sorry. Sorry. Oh? Sure. And Rob
Post would like to add a point.

DR. POST: Yeah. I’m not sure if this is on and
if everybody can hear me. Okay. In the discussion
yesterday, I just wanted to add a point, a suggestion,
for consideration in the discussion of dietary sources
of sodium and processed foods, and there are a couple
of points in the summary that Dr. Van Horn just
mentioned.

To consider that there is an issue in terms of
potential compromising of food safety, because a lot of
the substances that are used to promote food safety
that are antimicrobial in nature, anti-listeria, for
example, in certain processed foods are sodium-based,
so consider that issue in the discussion of sodium,
that suggestion.

DR. VAN HORN: Anything else? I don’t want to cut short any additional comments. Sure. Yes.

MS. McMURRY: Just one quick update, just to make sure you are all aware of another complementary effort at the Institute of Medicine. They have just recently convened a panel to look at strategies for reducing sodium intake to the 2005 Dietary Guidelines level; so to the extent that it’s possible, it would be nice to try to coordinate with that effort too.

DR. VAN HORN: Right. There were a couple of recommendations yesterday to look at linkages between this group and IOM, so thank you. Yes, that’s a great idea too, Kathryn. Thank you. Okay. Dr. Pi-Sunyer.

DR. PI-SUNYER: Okay. Well, good morning to you. Is this on? I am going to begin this discussion on energy balance and then Dr. Nelson is going to talk. She has been a member of the Physical Activity Task Force for the HHS that just finished their report, and so I’m not going to say much of anything about physical activity; she will. And then Dr. Christine Williams, who is a pediatrician, is going to talk about, a bit
about children and adolescents, and finally, Dr. Rafael Perez is going to say a few words about weight gain during pregnancy. And then we’ll leave it open for discussion.

Just, by way of background, I want to remind you of the trends and age adjusted prevalence of obesity. This is for adults 20 to 74, and you can see from 1974 up to 2000 there was an enormous secular increase in the number of people, who were obese, both males and females, and this continued pretty much right up to about 2006. Now, it may be flattening out a little bit. It’s not quite clear in the latest NHANES survey, but an enormous change in the population in terms of overweight and obesity.

You have all seen the CDC maps and they are graphic, and how they move from a very low rate of obesity to a rate where now up to three states are above a prevalence rate of 30 percent -- 29 percent, and growing rapidly around the country.

The same kind of trend you can see in children. This is data from NHANES for children showing you the same kind of increase over the same period of time, and
the children and adolescents, their rate has continued
to go up right up to the present time.

We are worried about this because of the medical
complications, particularly the ones you see on the
right; the coronary heart disease, diabetes,
dyslipidemia and hypertension. The paper today
announces that our rate of diabetes incidences has
doubled, so this is a very serious condition. There is
more data over the last five years; new data on the
relationship of obesity to cancer, and I’ll talk about
that a little bit; and then there is more data on non-
alcoholic fatty liver disease, which turns out to be
probably the greatest cause of cirrhosis in this
country after alcohol; and then quite a lot of data
related to pulmonary disease and sleep apnea.

So this really impacts Americans’ quality of life
and it really impacts the cost of medical care in a
very ominous way. We have not only the epidemic of
obesity, but this is related to an epidemic of what we
call the metabolic syndrome, which is really something
that leads to much greater risks for both Type 2
diabetes and cardiovascular disease. You can see how
the overweight and obese prevalence on the top -- men
in red; women in yellow -- is tracked by the metabolic
syndrome down below in white and blue. So, as people
gain weight, they also increase the number of them that
have the metabolic syndrome, which is clearly a risk
factor for both Type 2 diabetes and heart attack.

There is increasing data about the relationship of
obesity to cancer, and you can see here a study from
the European Commission -- showing you on the left,
men; and the right, women -- with the kinds of cancers
that have an increased incidence that is thought to be
related to increased overweight. In men, we are
talking about colon cancer, prostate cancer, kidney
cancer and gallbladder cancer; in the women, we are
talking about breast cancer, colon cancer, endometrial
cancer, kidney cancer and gallbladder cancer. So there
is, over the last five years there has been quite a lot
of new epidemiological; mostly observational -- really
observational data linking overweight and obesity to an
increased cancer incidence.

So what is the established science in this area?
Well, it’s really pretty simple. I think very few
1 people would argue that this isn’t true; the caloric
2 intake has gone up and physical activity has gone down.
3 The balance has shifted towards greater energy
4 reserves, and as a result, increased weight. There is
5 some argument around the world of how much is related
6 to intake and how much to physical activity, but I
7 think when we are dealing with the issue, we really
8 need to deal with both of them.
9
10 We know that you can change the way people eat and
11 the way they behave in terms of physical activity.
12 This shows you the data from the NIH Center in Phoenix,
13 which studies the Pima Indians showing you the Pima
14 Indians’ body mass index in Arizona and in Mexico; the
15 differences; the Arizonians have very high fat, high
16 calorie, high alcohol, a very sedentary kind of
17 lifestyle; whereas the Mexican Pimas were genetically
18 similar, are much more physically active and have a
19 much more traditional high fiber, high carbohydrate,
20 low-fat kind of diet.
21
22 In terms of the established science, we know that
23 obesity does lead to a number of major diseases. It
24 can also lead to a number of less prevalent, but
serious diseases. We know the public awareness of the
link between obesity and chronic disease is low, and
that the awareness needs to be increased sometime,
particularly in people who already have risk factors
for chronic disease.

The American paradox, I think in 2008 is that we
have an escalating trend towards poor nourishment and
health in a land of plenty, and because of the
sedentary lifestyles and poor food choices, many
Americans exceed their caloric needs without meeting
their nutrient requirements, and this was dealt with in
the last Dietary Guidelines Committee, and I think it
really needs to be dealt with in ours.

What kind of consensus do we have about all of
this? Well, first, it’s that we are making very little
impact; second, we don’t seem to have the adequate
tools to change lifestyle behavior in the way we would
like it to be changed; we need a lot more public
awareness of the relation of obesity to chronic
disease; and I think the awareness needs to increase
particularly in people who already have the risk
factors, which is a large part of the population, who
think they are healthy.

One question I think our Committee needs to address is should we focus on simply on prevention of weight gain, and should we avoid focusing on weight loss? The last Dietary Guidelines did talk a bit about weight loss. I think the really important thing from a public health point of view is to prevent people from gaining weight. It is very difficult to lose weight once you have gained it; you tend to gain it back. It’s sort of something that we have not been able to do well. On the other hand, I think if we focus the message on the prevention of weight gain, keeping people within the normal BMI categories, we might do better in the long run. This is taken from the old DRI. It seems logical to base estimated energy intake on the amounts of energy that need to be consumed to maintain energy balance in adults, who maintain desirable body weights; also taking into account the increments in energy expenditure elicited by their habitual level of activity. So, what we are saying is the energy intake estimated should depend on the energy required for somebody in a normal BMI category, and
also taking into advice their physical activity.

Now we know that small daily imbalances in energy intake have an effect in body fat mass. I am showing you this data from my colleague from Columbia, Michael Rosenbaum, which shows you what an excess intake of 12 calories per day, 25 calories per day, and 125 calories per day can do to change in body fat over one year. And you can see that it takes very little excess intake to really have a major impact on weight gain. And we know that Americans, as adults, continue to gain weight every year from age 20 to age 60. So I think the big message here is how we can get it across to people that with a small change in lifestyle they can have a big impact on preventing weight gain as they move from age 20 to age 60.

We have a sedentary lifestyle. This is from the Surgeon General’s report a while back, but it shows you that both in men and women, in all age groups, the percent not particularly participating in physical activity is extremely high, and this also we need to deal with.

What are the issues that need further discussion
or further evaluation? One, I think -- and these were actually dealt with, with the last Dietary Guidelines Committee, but I think we need to revisit them, because of new data in all of these. The first is what are the optimal proportions of dietary fat and carbohydrates to prevent weight gain; the second is how is physical activity related to body weight; the third is how much physical activity is needed to avoid weight gain; and fourth is caloric compensation different for solid and liquid foods?

Other issues, do energy-dense/nutrient-poor foods displace energy-poor/nutrient-rich foods, and does this lead to weight gain and nutrient inadequacy; and what kind of data do we have for saying this is true?

Other questions to address to prevent weight gain, I have already mentioned the optimal proportions of macronutrients; the effect of energy-dense foods; the effect of portion size; the effect of added sugars and the difference between added sugars in liquid and solids; the effect of snacks; and what intake pattern is most likely to prevent weight gain?

Finally, you know, what behaviors are most likely
to prevent weight gain? We have not been able to figure out how to change lifestyle behaviors to prevent this continuing increase in weight throughout the lifespan in Americans.

There are also questions we need to look at to prevent weight gain in special groups; children and adolescents, which Dr. Williams is going to speak about; pregnant and lactating women, which Dr. Perez is going to speak about; the elderly; and particularly, minority women, who have the greatest prevalence of obesity in this country.

There are questions about discretionary calories; do Americans really have any I think is the big question? And the second is, is it too difficult a concept? We wrestled with that a bit, you know, the last time around and I think we need to talk about whether this is a concept that, as a public health message, is difficult to get across. And the best way obviously, I think, to talk about it is in relation to physical activity, but we need to address that.

Potential guest speakers that I think would be helpful to us in our deliberations that I thought
about; one was Adam Drewnowski, who could come and
speak to us about nutrient density versus nutrient
adequacy; and Barbara Rolls, who has done so much work
on liquids versus solid compensation. So I think they
might be two guest speakers that we might like to hear
from.

So, in summary, the dietary factors that affect
energy intake that we need to deal with are nutrient
composition, energy density, portion size, liquid
versus solid, snacks, and then how physical activity
impacts the whole issue. So thank you very much for
your attention, and I’ll pass this on to Dr. Nelson.

DR. NELSON: Thank you, Xave. Well, I had the
wonderful honor of serving on the Physical Activity
Guidelines Committee over the past year. Also, similar
to this Committee, we had 13 members, if I remember
correctly, and a wonderful group of scientists; all
with expertise in physical activity and exercise, and
health and public health. And, I won’t go into the
whole history of why these Physical Activity Guidelines
came to be, but I wanted to talk mostly about sort of a
process and some of our main findings.
A couple of key things that I think are important are that, in terms of history is, when we look at the 2005 Dietary Guidelines, you can see that there is actually, you know, quite a bit of information around physical activity. I think that what was important was, because there is a whole -- I mean, really in the last 20 years there is really a real growth in the amount of research in the area and the number of scientists who have really devoted their careers, it seemed very important to actually have a committee that really could look at all of the evidence around, not just around energy balance, but around a number of different health outcomes; and to also put a little bit more information around physical activity beyond just the sort of 30 minutes a day of physical activity.

So, background, Americans are largely inactive; many opportunities for physical activity have been engineered out of daily life. I think what’s interesting is that if you look at the data around just leisure time physical activity, it’s been held pretty constant, but when you look at the number of sedentary activities and the hours of actual sedentariness,
that’s what’s really gone up. And that physical
activity is one of the most important steps people can
take for their overall health. So the process, we had
quite a -- this committee I have to say is quite
luxurious. We have a document from which to work from;
we have two years to do our work; we -- our committee
was convened with the first meeting, in June of 2007,
following the same FACA regulations. We met three
times. We met in June, December and February. We
reviewed the evidence from 1994, when the Surgeon
General’s Report came out, to 2008; and if we had more
time, our report would not have been 668 pages. I
think it would have been more like 350 pages, but -- so
our technical report, while it’s not printed yet --
it’s still on-line. It’s very accessible and I’ll give
you the link to that in a bit. So we submitted our
technical report in June, I believe, or May, late May
of this year, and then HHS had a writing group that put
together the actual Physical Activity Guidelines. And
I think that’s important for us to remember, you know,
we don’t develop guidelines. We look at the evidence,
So what’s new about the Guidelines? It was the first major science review in more than a decade to address Americans over the age of six in specific subgroups, and it really went beyond just the 30 minutes or more of most days of the week, although that was sort of a starting point for us; thus providing greater detail regarding dose and a lot more information around physical activities for Americans.

So, our major research findings, we were separated into subgroups that were looking at major chronic conditions, and what we saw was regular activity reduces the risk of most of the chronic diseases, as most everybody knows, and that these are some of the more salient findings that, in fact, some activity is better than none; basically that being sedentary confers the greatest health risk, and that any activity is better than none, but that there is added health benefits occur as the amount of activity increases; both aerobic and muscle strengthening activities are beneficial. Health benefits apply to people of all types, sizes and ages. Health benefits occur for
people with disabilities. Physical activity can be done safely. Benefits far outweigh possible risks. Physical activity provides health benefits regardless of body weight changes over time, so at any given body weight, a person who is more active is going to be healthier than a person who is not active.

In terms of the sort of dose of activity, a total of two-and-a-half hours a week of moderate intensity aerobic activities substantially reduces the risk of many chronic diseases and other adverse health outcomes. So, this is really back to the 30 minutes a day. I mean, that’s where the evidence seemed to really converge now. I will tell you that some of the issues -- maybe I am speaking personally here -- but, because we have had this guideline for so long, most of the good research studies really are designed to look at this dose of physical activity. So, I mean, one of our research sort of questions is, you know, is there a range of doses that confer; but this is what sort of settled out. Again, any activity is better than none, but this is really where most of the health benefits confer, and then as people move from two hours and 30
minutes a week towards five hours or up to an hour a day of moderate activity, you get even more benefits; really basically more is even better especially if you have some health concerns.

And then the other thing that I think is important is that -- oh, wait a second -- I just wanted to go back -- what I don’t have here is in fact that -- well, I’ll go with the Guidelines -- just a second. So, when we look at children, and this is really -- there are no great changes from what the Dietary Guidelines did in 2005. You are all looking at similar data. But, with children, one hour or more physical activity a day that’s at least moderate is beneficial. Most of the one or more hours a day should be either moderate or vigorous, and to do vigorous intensity physical activity at least three days a week, so that children should have some vigorous activity. As part of one or more hours daily activity includes some muscle strengthening activities at least three days a week, and as part of one or more hours of physical activity include bone strengthening activities at least three days a week. And the real key here is that we need to
encourage young people to participate in physical activities that are age appropriate; that are enjoyable and offer a variety of activities. We are not talking about sending the eight-year-old to a gym and lifting weights. We are talking about playing on the jungle gym and climbing trees, and doing hopscotch and jump rope, and everything else that young people at least used to do.

In terms of adults, 18 to 64, similar two hours and 30 minutes a week of moderate intensity or one hour and 15 minutes of vigorous intensity activity, or a combination of the two.

So this was the data. We weren’t sure where this was going to show up, I’ll tell you, but it was a charge right off the bat that we decided we wanted to look at was this -- is it intense activities or is it moderate activities, and then we weren’t sure, and in the end, it can be either or. And I think this was, from a public health message, I think it was really important, because it shows that if you want to spend less time, you can do it more intensely. So, it means that it makes it a little easier for those of us that
have challenging jobs to get some of these, meet some
of these deadlines. And that -- or these
recommendations. And that muscle strengthening
activities involve all major muscle groups should be
performed on two or more days of the week, and there is
a variety of ways to get those activities as well.
Additionally, five hours a week of moderate intensity,
or two-and-a-half hours of vigorous activity, or an
equivalent combination for even greater additional
health benefits, especially for people with some
chronic conditions, and especially with issues around
weight control.

So with older adults, the same exact guidelines as
for adults, but when not possible, be as physically
active as someone’s ability and conditions will allow,
and this goes all the way up through the oldest of old.
And do exercises that maintain or improve balance, if
the person is at risk for falling. There is some very
good data for individuals who are at risk for falling
for reducing falls. And those without chronic
conditions and symptoms do not need to consult a health
care provider about physical activities. So this
barrier for older adults, especially that they have to see a physician, that we didn’t see any evidence that was absolutely necessary.

So some additional considerations, other subgroups of the population and Physical Activity Guidelines included persons with disabilities. Jim Rimmer, who was fabulous, really took this on, talked about a lot of small research studies that are out there, but really compiled a lot of good evidence, and that women during pregnancy and post-partum period, the guidelines for them are really no different; however, they just have to monitor themselves. And that adults with select chronic conditions, especially with arthritis and osteoporosis, we dealt with as well.

So a little bit about sort of -- I have to say, we spent a lot of time on the weight control issue. What I will focus on for just a second is the bottom line here; is that the one thing that we, you know, it’s -- I don’t want to say, duh -- but, it was so important is that you really cannot look at physical activity and weight control unless -- without considering dietary intake and energy intake. And so, what we did and what
I hope this Committee will do also is we really said that you have to consider nutrition; and in fact, if the data show us that if you just add physical activity to someone’s life, they don’t lose weight because they end up compensating by eating a little bit more, so you really -- there has to be a dietary intervention when you are talking about weight loss. So, in terms of -- we separated things out into three different categories; weight stability; weight loss; and weight stability after weight loss. And the data on weight stability -- and, Xave, I think this will be the big challenge for us and I don’t think we need to look at this data again, because we just have -- we can refer back to the report -- but that weight stability over time, it’s really almost impossible to do a well-designed, randomized controlled trial, because you are talking about trying to measure no change, and you are talking about large groups of people, but the preponderance of the data when we looked at it, it’s somewhere in the vicinity of 150 minutes of moderate activity or 75 minutes a week of vigorous activity is helpful for people in terms of weight stability, but
again, you can’t isolate it out of dietary intake. In terms of weight loss, we are talking about larger amounts of physical activity; and in terms of weight stability, it’s even more physical activity. But again, this is where you need to tie the dietary intake together with that.

So the sort of -- the theme here is be active your way. I think that one of the important parts is that there is a variety of ways to get physical activity into your life; be active, healthy and happy. The mental health outcomes here were also great. The website is listed here. A couple things I want to say is, first, I really want to thank Rick Troiano, who is in the audience today, because he was the one who really put these slides together. I just edited them. Our whole Committee was provided with slides around the process, so don’t think that I was so clever as to be able to put these all together.

But, the other thing, a couple of things that I think were important potentially for this Committee as we go into sort of what I would call an evidence-informed process, there was so much different data that
we looked at for our Committee, and one thing that we did -- and you can look at it later -- but, in the summary chapter, you have it in part E, page actually E-2, this is in section 4 of your binder, we put together a table, which was a way so that we could, across all of our subcommittees, categorize the kind of science, the research that we were looking at. So we weren’t individually coming up with qualitative ways of discussing our research. We -- this was - this sort of comes out of more of the pharmaceutical or heart disease kinds of way of looking at research, but we sort of massaged it a bit so that it could be used for physical activity, and I think we could also use it around nutrition. It’s table E-1. So we are looking at the type of evidence, and we categorized the evidence into four different types of evidence, and then we looked at the strength of the evidence.

So, in fact, for example, Type 2 could be randomized controlled trials or meta analysis with important, with some limitations or non-randomized clinical trials. Type 1 is randomized controlled trials without major limitations. Type 3 is well-
designed prospective cohort trials or other observational trials. And Type 4 is much more around sort of anecdotal evidence or expert opinion, and then the strength of the evidence either is strong consistence across studies; moderate or reasonably consistence across studies; or weak or limited inconsistence across studies and populations. And this was a way for us to categorize the research across subcommittees, and I would hope that we could possibly do a sort of thing as this. We spent a lot of time around this, because again, there was just so much different types of data that we were looking at as just an idea.

So, I am happy during the discussion to answer more questions, but I really -- I think it will be important -- I don’t believe that this Committee needs to re-evaluate all the physical activity data over the last 14 years, because we have just done it and the report -- there is a lot of information in the report, and I’m happy to be that link between the two committees.

DR. VAN HORN: Excellent. Thank you.
DR. WILLIAMS: Thank you very much. In my presentation this morning, I would like to address some issues related to childhood obesity and energy balance.

We all agree that children need a high quality diet that’s sufficient in energy and nutrients, as well as adequate daily physical activity, in order to maintain health during childhood, and also to prevent risk of future chronic disease.

One of the most disturbing public health trends in the past few decades, however, has been the increase in obesity in youth. Although the increase in obesity has also affected U.S. adults, and indeed many populations around the world, the magnitude and rapidity of the increase among U.S. children and adolescents has been significant enough to label it an epidemic.

Since the late 1970s, the prevalence of obesity has increased more than 100 percent in children six to 11, and 200 percent for adolescents 12 to 19. It’s generally accepted that both genes and environment contribute to obesity risk, but since the increase in prevalence of obesity was too rapid to be explained by genetic drift, the consensus is that it’s the result of
a shift in energy balance.

Since Dr. Nelson addressed issues related to physical activity and obesity, I’ll focus on some changes in dietary intake of children over the past 25 years; changes that may have contributed to energy imbalance. A closer look at the prevalence rates for childhood obesity reveal the changes since the 1960s; in the beginning, the slow and almost imperceptible increase before 1980, and then the rapid increase over the next two decades. Here in bar graph form is the same data. You will note that there is some overlap in the dates for the most recent surveys depending on which time points were included in the analyses and reported in the three most reports in the JAMA articles; 2004, 2006, and most recently, in January 2008.

When we take a closer look at the changes in the prevalence rates, there may actually be three time periods of interest. Following the first period of very slow increase between 1963 and 1980, there was a period of very rapid increase between 1980 and 1999. Since then there is some recent evidence that perhaps
the epidemic has begun to slow down or even begun to
decrease. During the most recent period, there seems
to be a change. When the prevalence rates for 2005 to
2006 were examined, they found that they were not
significantly different from the rates for 2003 or
2004, so that they were combined. There was actually a
small, but non-significant decrease in overall
prevalence of overweight for 2 to 19-year-olds; from
17.1 percent in 2003 to 2004, to 15.5 percent in 2005
to 2006, and this was somewhat exciting, because up to
this time rates had just continued to increase. The
decrease, however, was seen only among non-Hispanic
white youth and not among minority children.

More about those recent changes. During 1999 to
2002, and even through 2003 and 2004 it appeared that
the prevalence rates were still increasing, but now
that the latest figures for 2005 and 2006 have been
added, there is a suggestion that perhaps a change has
begun and the epidemic is beginning to level off. In
fact, analysis of prevalence rates showed no
significant trends between 1999 and 2006. The authors
cautions, however, that data from 2007 to 2008 will be
needed to further examine the trends.

So I think it's interesting to look at changes in children’s diet relative to the time periods when obesity rates were increasing very slowly, and then during the periods of rapid increase between 1980 and 1999; and eventually to look at dietary changes in the last seven years, between 1999 and 2006, when rates may have leveled off, or there is at least a suggestion of that.

Have there been measurable changes in energy intake in children and youth since the early 1960s? What happened to children’s dietary intake between 1980 and 1999 when the -- during the period of most rapid increase in childhood obesity; and are there any recent healthful dietary trends that may be contributing to a slow down in the childhood obesity epidemic?

The question about energy intake among children; what do the national surveys show, and there is a lot more data that can be added to this chart, but if you look first at the, some of the data from the most recent surveys, 1999 to 2005 and 2006, it looks as if the energy intake is fairly stable during this time.
The previous analyses show mixed results; Troiano compared energy intake between NHANES I and NHANES III, and the only change noted was an increase for adolescents, especially adolescent females. But it was difficult to explain this since the increase in obesity over that time had occurred in all age groups, not just teenage girls. (Inaudible) compare energy intake for children between 1977-1978 and 1989-1991; 1994-1998 surveys. In this comparison, total energy intake had increased with similar changes for children of all ages. They also reported shifts from at home to away from home food consumption, and from meals to snacks. There are, of course, a lot of methodologic difficulties in assessing dietary intake in children, especially with collecting proxy data from parents and caregivers.

Here is a closer look at some of the data between CSFII 1989-1991 and 1994-1995. Aside from looking at energy intake, what about changes in children’s food patterns and food choices since the late 1970s? Just briefly, when the dietary intake of children in 1977-1978 was compared with recent intake in NHANES 2001-
2002, some key changes were noted; beverage preferences have changed significantly; children and teens who consumed fruit juices, fruit drinks and ades and soda were drinking more of these beverages in 2001-2002 than they did in 1977-1978. Higher fat food choices had also increased. For the beverages, the changes over the past 25 years since 1977-1978 and NHANES 2001-2002 for children six to 11, milk has decreased from 61 percent of total beverage intake to 33 percent; soda has increased from 15 to 33 percent of total beverage intake; and for teens 12 to 19, soda replaced milk as the beverage of choice; from 1977-1978, milk made up 51 percent of all beverages by gram weight, while soda made up 29 percent. By 2001-2002, this was reversed; soda made up 50 percent and milk 23 percent. The majority of soda, 95 percent consumed by children and teens is regular soda. Fruit drinks increased slightly 14 to 20 percent, and fruit juice increased slightly also 10 to 14 percent in children; 9 to 10 percent in teens. Again, if you look at beverage intake among children and teens, you can see the decrease in milk and the increase in other beverages.
Another recent change between 1977-1978 and 2001-2002 has been that higher food choices have increased for U.S. teens and children. Consumption of pizza, tacos and snack food increased dramatically for children and teens between -- in these 25 years. There were large increases in mean intake of savory grain snacks, pizza, Mexican dishes and candy. There was a sizable increase also in fried potatoes, but a decrease in intake of vegetables.

So, in general, over the past 25 years that roughly coincides with the rapid increase in childhood obesity, food and beverages choices have changed considerably. Beverage choices shifted from milk to less desirable choices; those which typically have higher caloric content relevant to nutrients they provide showed large gains in popularity.

Another major trend during this time was a trend for children to eat more food away from home. Even for two-to-five-year-old children, food consumed at home decreased from 88 percent to 76 percent; and food away from home -- food consumed at home decreased, and food away from home doubled from 12 to 24 percent, and those
are for preschool children. This is even greater for
teens 12 to 17, where foods eaten away from home
increased from 20 to 35 percent over the same period;
and for six to 11-year-old children the increase was
similar, 21 to 32 percent. The problem is that meals
consumed in restaurants are not as healthy as home
meals. They are lower in total unsaturated fat and
sodium and higher in calcium and iron. Children also
are snacking more. I’m sorry -- lower in calcium and
iron. Children are also snacking more, tending to
replace meals with snacks. Young children gradually
increased the number of snacks they eat each day from
1.73 to 2.29, so the total energy increased because
children are snacking or eating a greater number of
snacks each day. The snacking patterns of two to 18-
year-old children also increased. More children are
snacking now. The average energy intake from snacks
increased from 450 to 600 calories, and the energy
density of children’s snacks has also increased.

So the question now is, if the prevalence rates
for childhood obesity are beginning to slow down, have
food patterns changed since 1999 to 2000? Have there
been any improvements? Have there been any changes in energy intake, as well as macronutrient/micronutrient intakes since 1999; or diverging difference among children by race and ethnicity, have choices of beverages changed again? Have the amount of beverages changed recently? Have higher fat food choices decreased? Has there been a change in patterns of eating out or purchasing take home foods? Have economic hard times encouraged more at-home meals? Are children and teens more physically active than they were in 1999? We need a lot more information on these questions, but it’s likely based on current knowledge, especially energy balance.

The 2010 Dietary Guidelines will need to emphasize key issues that are most likely to help children, adolescents replace some current food choices with more healthful options, including fruits, vegetables, whole grains and calcium-rich foods and beverages. This will be critical in helping children meet nutrition, nutrient requirements and reduce risks of future chronic disease. Thank you.

DR. PI-SUNYER: Thank you, Dr. Williams. Now Dr.
1 Perez is going to speak briefly.

2 DR. PEREZ-ESCAMILLA: Good morning. I will talk
3 about recent developments related to gestational age
4 weight gain; maternal and child obesity-related
5 outcomes.

6 In 1990, the Institute of Medicine came up with
7 the actual recommendations for gestation and weight
8 gain, and brought down these recommendations based on
9 pre-pregnancy body mass index. In 1990, the criteria
10 that the IOM Committee used was mostly related to the
11 prevention of small-for-gestational-age babies. And,
12 research by Barbara Abrams and others has shown that
13 these recommendations do work, a very reasonable
14 prevention of small-for-gestational-age weight
15 babies.

16 The problem that we are facing now is that we are
17 now in the midst of a major obesity epidemic that has
18 affected women, to a large extent, and in particular,
19 low income minority women; such as African-American and
20 Latina women.

21 As you can see from the recommendation, in 1990,
22 the Committee decided to recommend that among women who
were obese before pregnancy that they should gain at least 15 pounds. Unfortunately, there wasn’t a lot of evidence based behind this recommendation, and this recommendation caused -- I mean, has caused an enormous amount of confusion with some coming up with their own recommendations claiming that women should not gain more than 20 pounds all the way to some groups claiming that women should gain less than 15 pounds if they were obese or if they are obese before pregnancy. Because of the context of the major obesity epidemic in which we are, the IOM decided to call for the formation of a committee that is currently reviewing the gestational weight guidelines, and this report is expected to be released in 2009.

However, there are materials that can be shared now, because our AHRQ published an evidence-based systematic review on maternal and child consequences of gestational weight gain that is available at this website and that systematic review was released just a few months ago, so, it is very updated.

I have identified two key issues that I want to share with the Committee today, and the first one is
that it is clear that high pre-pregnancy body mass index is a significant predictor of excessive gestational weight gain and maternal post-partum weight retention and that, in turn, is a significant predictor of serious risks for chronic disease among women. Excessive gestational weight gain independently of pre-pregnancy BMI is a significant predictor of post-partum weight retention, which is a major cycling issue that eventually leads to major obesity problems among women that have multiple pregnancies. High pre-pregnancy body mass index and excessive gestational weight gain are definitely associated with the delivery of large-for-gestational-age newborns, and being a large-for-gestational-age newborn, in turn, increases the risk of childhood obesity, insulin resistance and Type 2 diabetes later on in life. So, this issue of gestational weight gain during pregnancy and maternal pre-pregnancy BMI has major implications for maternal health, and also for the childhood obesity epidemic. The implications of this, first the issue is that the fetal nutrition and hormonal milieu is likely to affect the risk of childhood obesity and chronic
disease perhaps via epigenetic mechanisms. Secondly, preventing maternal obesity before pregnancy is likely to have a positive impact on both maternal and child chronic disease outcomes. And lastly, I want to mention that many questions remain regarding the safety of promoting weight loss during pregnancy, because with that type of approach we have to take into account the health of the mother, but also potential risks for fetal health if the mom enters a ketogenic status.

The second issue that I want to mention is that the AHRQ report fully confirms the 1990 IOM findings regarding inference of maternal underweight before pregnancy, and to what the maternal gestational weight on risk of small-for-gestational-age babies. SGA -- since then, however, evidence has been strongly accumulated that SGA is associated also with an increased risk of childhood adiposity accumulation and risk of type II diabetes and cardiovascular later on in life, and there is a good number of studies that have been published since then along the themes of fetal programming or developmental origins of adult health and disease. And I want to emphasize that the risks
for linking SGA with childhood adiposity and chronic disease later on in life seems to be strongly modified by the growth rate during infancy. So I know that the Dietary Guidelines are supposed to look at the U.S. population after two years of age, but it is very difficult to deal with a childhood obesity epidemic if we don’t deal with the infant nutritional, early nutritional issues as well.

So, the implication of this key issue is that preventing maternal underweight before pregnancy and to what amount the gestational weight gain are likely to also have a positive impact on preventing childhood obesity and subsequent chronic disease outcome. So, at both ends of the spectrum, SGA or LGA, they both appear to have strong implications for the childhood obesity epidemic.

And last, but not least, it is my hope that the 2010 Dietary Guidelines that impact maternal intake and physical activity during pregnancy should take fully into account the gestational weight gain IOM report that is due next year. Thank you.

DR. PI-SUNYER: Thank you, Dr. Perez. Linda,
that’s it. We are open for discussion.

DR. VAN HORN: Excellent. Well, thank you, everyone. Those were wonderful presentations and I know there will be a rich discussion following all of that. I don’t know how you’d like to begin. We can just open it up and take any of the presentations, or do you want to go in order?

DR. PI-SUNYER: I think anybody who wants to ask questions or make comments, we’d love to have them. Larry?

DR. APPEL: Yes. Those were great presentations.

A comment --

DR. VAN HORN: Oh, please say your name before you speak.

DR. APPEL: Larry Appel.

DR. VAN HORN: Thank you.

DR. APPEL: I have a -- I would propose a question that you consider, at least on the list, and that is, you know try to make this action-oriented. In terms of behaviors that we use in our weight loss trials; self-measurement of weight; the self-measurement of physical activity; calorie counting and their effects on
preventing weight gain or encouraging weight loss might be questions that, you know, could be answered that would actually have an impact on what people do and at least put those on the list, I would hope. And I also wanted to toss out a concept -- you know, I am sure you thought a lot about the 2005 Guidelines, where we focused on weight and we didn’t use calories as an outcome variable that would drive decisions, and you know, I have thought a lot about this. You know if, as you point out, the difference in calorie intake that accounts for the obesity epidemic is 100 calories per day, and if it’s multi-factorial -- let’s say there are 10 factors, each accounting for 10 calories per day -- do we have the tools, both epidemiologic -- primarily epidemiologic, to discriminate at that level; and instead of saying, we don’t have the evidence, we might want to ask, will we ever have the evidence for, let’s say, sugar-sweetened beverages or portion size, and if we don’t think we are going to have the evidence, then I think we need to make decisions about whether to act.

DR. PI-SUNYER: Yes. Well, I would agree with you. I think it’s very difficult when you get down to
under 100 calories to really be able to measure it and, you know, so the evidence isn’t really there except in short interventional studies.

DR. APPEL: Yes. And we sort of punted saying, well, let’s get the evidence, but if it can’t, if it never will occur, we might have to make some decisions.

DR. SLAVIN: Yes. This is Joanne Slavin. I am on this committee too, but I just want to talk about the Jim Hill stuff, where you look at people that actually are successful and calorie counting and exercising that’s what they do. So I think we have -- it’s not, you know, exactly the kind of research people are looking for, but it’s very clear that if you want to be successful -- and, you know, I always talk about that -- I give the example that french fries at the state fair is 7,000 calories and people eat those french fries. And I say, you’ve got to walk to Eau Claire, it’s 70 miles, so think about. You know, I think linking it is critical to people; there is a price to pay. You might want to eat it right now, but down the line is was it worth it?

DR. APPEL: Yes. This also has implications for
calorie labeling widespread. You know, if what you people are asked to do; to monitor their weight and monitor their calories, and if they are eating out frequently, you actually have to provide calories for those people who are actually calorie counting.

DR. VAN HORN: I’d like to just jump in on that one as well. We were discussing this, Joanne and I, on the way over here, and the whole concept of calories I think is something that we might want to embrace at this point. I personally have heard over the years, well they are too hard to calculate; you know, people won’t want to do the math, I mean all of those kind of statements, and I think we have now perhaps a more sophisticated society that, if they can learn to know their cholesterol number they might be able to learn to know their calorie number and what they are supposed to have and how to get them, and how to burn them. So, I would just like to add to that concept of you know perhaps starting to get people thinking more about the energy balance using something more objective, like calories.

Sorry. Go ahead.
DR. PEARSON: I think -- this is Tom Pearson -- all of you dealt tangentially with the issue of this paradox of, as education and income levels go up the obesity levels go down and this apparent competition of food insecurity programs on the one hand and over nutrition on the other. So I guess one of the questions I had is that there -- is there a literature and evidence base to look at the issue of if you attack the obesity issue in one way, you don’t affect the under nutrition issue in another way? We are getting into some economic difficult times. These are some times in which you are going to have larger portions of the U.S. public in need of some food assistance, and so what you have at the same time of this co-existence of the obesity epidemic somewhat of a historically and unusual situation in which you are going to have two mega trends coming together where you want to actually want to reduce consumption at the same time as you really historically are going to be looking at people who are really nutritionally and adequately taken care of. So I guess the question is, what is the evidence base that you can actually do both of those
1 successfully?
2 DR. PI-SUNYER: Well I think that we can certainly
3 look at that. Dr. Post, do you have any thoughts on
4 that, in terms of the U.S. Department of Agriculture
5 and their programs, and you know, what happens to a WIC
6 woman when she gets pregnant and is getting WIC food?
7 Does she gain more weight when she is pregnant, or do
8 we have any data on things like that?
9 DR. POST: There are some data. We could organize
10 that for you in the next, in the interim certainly and
11 get that to you and this subcommittee, in terms of what
12 we do have.
13 DR. PEARSON: Yes.
14 DR. POST: Working with the food and nutrition
15 source.
16 DR. PI-SUNYER: Because I think it’s an
17 interesting question. I have often thought that we
18 could solve the obesity problem if everybody went to
19 college and got a certain minimum income.
20 DR. APPEL: You may be right.
21 DR. NELSON: But not so much anymore, I don’t
22 think.
DR. VAN HORN: Rafael?

DR. APPEL: It would be easier.

DR. PEREZ-ESCAMILLA: I just want to mention, you know, the food insecurity problem in the U.S. is mostly related to dietary quality and not to quantity, and that in fact, at least among adult women, there is really consistent evidence that food insecurity is related to obesity, not to under weight. So, that’s a context in which food insecurity is happening in the U.S.

DR. FUKAGAWA: Two comments. I was really -- this is Naomi Fukagawa -- really heartened by the trends in pediatrics and seeing that it potentially is leveling off, which means that somewhere we have been effective at teaching and enforcing the message in at least the age group that is amenable to changing their behavior.

One of the questions that I have is, do we have any data as to how the general public where this epidemic is occurring, putting aside you know the concerns about the pediatric age group, as to whether or not they perceive it as a problem? You know, we are telling them that obesity is an epidemic; it’s going to
hurt them; you know, there health is going to go
downhill sooner rather than later, but --

DR. PI-SUNYER: Well I think this relates to Dr. Pearson’s point.

DR. FUKAGAWA: Yes.

DR. PI-SUNYER: I think a lot of people, particularly some minority groups and some lower socioeconomic groups have higher, other priorities that are much more -- that are pressing on them much more than their weight, and so they may have some sense that 15 years down the line they might get diabetes, or 20 years down the line they might get a heart attack, but that’s a long way away and right now they have all these pressing issues, and so the priority is not as high, you know, as maybe it should be in terms of their future health.

DR. WILLIAMS: Chris Williams. I’d also like to say that working in the clinical field there is always a lag between the time that materials are available for counseling minority families; and especially in Spanish and other languages. Working with minority families in the area of Columbia, you are always struggling to find
materials, because there is a lack of materials for families like that.

DR. PI-SUNYER: Again -- excuse me -- in New York, you know, in the public school systems there are 187 languages that are being, as second languages, that are being taught.

DR. VAN HORN: Shelly.

DR. NICHOLS-RICHARDSON: Sharon Nichols-Richardson, just in response to Tom’s question. The data that I presented yesterday was taken from the reports on the food stamp program and the school lunch program, and those data were broken down into the different groups. And, if I had to boil that down to a single message, there is not much difference based on participation versus non-participation versus a higher SES status, in terms of diet quality, related to the nutrients that were presented yesterday.

DR. ACHTERBERG: Cheryl -- Cheryl Achterberg. I just wanted to toss in the comments too that I think the Committee should look at the data around food variety, and I know that’s hard to measure, I know it’s difficult, but look at it in the context of these
eating patterns that lead to weight gain or not lead to weight gain, especially in terms of the development of childhood obesity. You know, is it a function, in other words, of a harried mother coming home and just throwing a bag of chips at a child and the child eats the whole bag and that’s the extent of variety, or if in fact they do consume more variety, is that associated not only with nutrient adequacy, but ultimately a lower calorie intake?

DR. NELSON: This is Mim Nelson. I really wholeheartedly support the self-monitoring piece. I think the data is so strong, and it’s not just from the, you know, the Jim Hill’s work. So, I really wholeheartedly support that.

Two other sort of comments. Xave, I am not sure about this linking sort of discretionary calories with physical activities, and I’ll tell you why. Because most of the research that we just looked at, when you -- and it could be -- there may be a message in there that we could use, but the problem is most of the research shows that when people increase their physical activity by 20 minutes a day or 30 minutes a day they
I don’t lose any weight. So, we just have a natural propensity to eat a little bit more to maintain whatever body weight we have. It may help with not gaining weight, but I think that we may be overstating what the research says about if we just increase physical activity a bit, we’ll be okay. So, I am a little bit concerned about -- because we just naturally increase our energy intake, although certainly we need to promote physical activity. But the other is -- and this gets back to I think some stuff that Larry said yesterday about some of the cultural, you know, differences that are out there, other cultures. I mean, I am thinking of Japan, and they have this sort of cultural saying, hara hachi bu that is around eat 80 percent of what you need. You know, try to stop before you are 100 percent full. And I think that it’s really indoctrinated into the culture about you just don’t eat until you are full, and I don’t think -- I wonder if there are ways to -- you know, when we do weight control studies or weight loss, we talk about trying to just eat a little bit less than you need. And that -- it might get to calories, but basically we have to eat
less, and that’s I think a message and I think there is some research to back that up that’s very important, so.

DR. PEREZ-ESCAMILLA: Can I go back to Naomi’s question, because I think it’s a very important one. You know, my research group has done extensive work with the Puerto Rican community in Connecticut, and this is a community that has been decimated and continues to be decimated by the Type 2 diabetes epidemic, and we have found that they are very aware, the vast majority of them, that it’s not a good idea for their kids to be obese and that that is indeed a risk factor for Type 2 diabetes. But, as it was mentioned by Xavier before, there are other priorities, there are major barriers that keep them from doing the things that need to be done for their kids to not be obese. And you know some may be difficult choices, but a lot of them are structural system level type of barriers; and if we don’t understand what those barriers are, it’s very difficult to make progress in addressing childhood obesity in these communities. But, it is not that they think it’s great for their
1 kids to be obese.
2
3 DR. NELSON: Can I just respond? Because --
4 IOM has just convened a committee on looking at
5 community factors for childhood obesity prevention, and
6 I’m thinking a lot about some research that a close
7 colleague of mine at Tufts is doing around really sort
8 of environmental interventions, and it’s really so that
9 choice is not an issue; it’s really that the community
10 is less obesogenic, and it works. And so I think that
11 there are ways that hopefully the policies that come
12 out of this could help with sort of community action.
13 I am thinking about the Physical Activity Guidelines
14 Committee and I show this lovely brochure -- you can
15 see I am doing a big ad for our -- but this is a, you
16 know, a community guide for trying to figure out the
17 built environment, connectivity and things like that,
18 and I really like to think -- I mean, Xave, you brought
19 some other, you know, it’s not just about what we eat,
20 but it’s also how we eat, and so if we can sort of
21 think creatively about that so that the parent isn’t
22 having to make these choices, it just naturally is
23 healthier for the kid to be there. I think there is
good data now and I am happy to share some of our
current research on these sort of environmental change
interventions that work around nutrition and physical
activity.

DR. VAN HORN: Okay. I think we should be moving
along, but in keeping with the pledge I made yesterday
and hearing about all this physical activity, I want
everyone to stand up for two minutes, just shake
yourself out, take a deep breath, stretch, do whatever,
and then we’ll start on carbohydrates.

(Whereupon, at 9:49 a.m., a brief recess is
taken).

DR. VAN HORN: All right. Next, we are going to
be talking about the role of carbohydrates on health
and Dr. Slavin is going to address this topic, and she
pretty much has got the show on this one. So, go for
it.

DR. SLAVIN: Thanks, Linda.

DR. VAN HORN: Yes.

DR. SLAVIN: All right. My outline -- what I was
told to do, and I am from Minnesota so we are very
compliant -- we just follow the rules -- was to first
summarize the recommendations in the 2005 Dietary Guidelines relative to carbohydrates; do a little bit on the background and then what new information has happened since the 2005 Dietary Guidelines that we’ll need to put into our review and consideration.

So, Dietary Guidelines for Americans 2005 choose fiber-rich fruits, vegetables and whole grains often. I don’t think there is -- I’ve heard much disagreement to that. Choose and prepare foods and beverages with little added sugars or caloric sweeteners, such as amounts suggested by the USDA Food Guide and the DASH Eating Plan. I am going to talk some more about what has been happening since that point. And then the third one; reduce the incidence of dental caries by practicing good oral hygiene and consuming sugar and starch-containing foods and beverages less frequently, which is also a known and could easily flow right into the 2010.

All right. Some other things in the 2005 carbohydrates part of the healthy diet; positive association between consumption of sweetened beverages and weight gain -- a study that had come out in 2004,
I’ll talk a little bit about -- reduced intake of added sugars may be helpful in weight control and achieving recommended intakes of nutrients, and this is kind of the discretionary calories idea that if you don’t have any discretionary calories there is not going to be much room for the sugars; and then to reduce the risk of coronary heart disease and promote laxation recommends intake of 14 grams of dietary fiber per thousand K-cals, which came out pretty much directly from the DRIs.

All right. I’m going to do a little background and I have to thank my friend, George Fahey. We do this talk together and he and I have put it together, so some of it may have come from him, so I thank him for that.

Looking at carbohydrates we usually talk about chemistry. And remember too, carbohydrates are done by difference, so even though carbohydrates I think are really important, it’s half our calories, very few people -- you know, measurement -- there are a lot of issues. But, from a chemical perspective, we have monosaccharides, which really don’t occur in foods
much; sugar alcohols, which we add to food; disaccharides, you know, lactose, sucrose, which are the sugars we eat; tri’s, which are pretty uncommon; oligos, three to ten glucose or carbohydrate units; and then polys are bigger than that. So it’s mostly this chemistry idea, which is good, but it doesn’t help us much when we get into dietary guidance. So, when we look at the nutritional perspective, what else can we talk about with carbohydrates? Do they get absorbed? Do they -- you know, do they get into the body? Are they digestible? The bigger ones, anything past a monosaccharide if it’s going to get in, it needs to first be broken down so we can get it in. Once it gets down to the large intestine, the formidability of carbohydrates. And then there are certain fibers or other non-formidable carbohydrates that nothing happens; they pretty much goes straight through. So, if you think of eating sand, if you have a baby in a sand box, it comes through and there are some carbohydrates that end up in the diaper. Nothing happens. They just go along for the ride. Absorbable carbohydrates that don’t require
digestion are the monosaccharides; glucose, galactose and fructose. They don’t occur naturally in foods so they really aren’t any of those, or they are very small amounts, in general, very small amounts. A little bit of fructose. We add fructose to foods, so there is going to be some, but typically those are not -- there is not much of that naturally occurring in foods, so there is not much exposure to that. Digestible carbohydrates are the ones that get enzymatically digested. So, you’ve taken lactose, it gets broken down. Sucrose, the same thing. And remember, you know, lactose, you know, the components of lactose; glucose, sucrose, fructose, glucose, and then just maltose being the disaccharide of starch. And that’s most of what -- the carbohydrate exposure we have is starch; most of what we eat typically.

The whole thing of fermentable are ones that get down to the large intestinal tract -- so lactose and sucrose perhaps sometimes. If you are lactose intolerant, they definitely get there. Pectin, some of the other fibers; beta-glucans; psyllium; gums; oligosaccharides, those three to tens; some of the --
you know, things that are in beans, onions that cause intestinal gas; and then just resistant starch. So starch can -- you know, as long as you digest it and absorb it, you get the glucose. If it’s resistant to digestion, absorption, it gets down to the large intestinal tract, functions similar to fiber. Then there are some of these celluloses hemicelluloses. There are some resistant maltodextrins that nothing happens. They really do go in and come out. And, you know, they may absorb some water along the way, increase stool weight, but really nothing else happens.

This is another big thing. I wanted to just talk about glycemic versus non-glycemic, and up on the top -- a little hard to see here -- but glycemic, we talked about the free sugars getting metabolized by the liver. There is some thought on lipid metabolism in fructose. A lot of that is in animal studies. Maltodextrin, you know, these starch components and they are either rapidly available or slowly available, and we’ll talk more about that; how important that is to glycemic index.

All right. And then the whole non-glycemic and
the fiber committee dividing it into total fiber; both
dietary fiber, which is naturally occurring in foods;
and functional fiber, which is isolated fibers and
those varied a lot. So, you know, if you look at total
fiber, they are very different compounds, so -- but the
dietary fiber is a food matrix, so it’s actually food --
you know, it has to be in food for it to be dietary
fiber. Functional fiber is just fibers we put into
food. Go down the way, oligo saccharides, they can
both be intrinsic, so if you look at wheat, onions,
there is a lot of oligo saccharides naturally
occurring; beans obviously -- you can think of things
that cause intestinal gas, and that’s them. And we can
also add those to foods. So oligo saccharides can be
added to food. And then just sugar alcohols, another
food additive that functions similar to these, you
know, they can get fermented and cause intestinal gas
and other problems. So there is a lot of exposure to
carbohydrates, and this is a slide which just tells you
that everyone is really different.

So chemical identity we talked about already; the
food matrix, how it’s put together, and then just how
it’s consumed in other foods, obviously meal factors, other properties of the carbohydrate, individual variability, and everybody’s gastrointestinal tract is different, so we do see some differences there. But, it’s either going to get absorbed in the small intestine and then we are going to get glycemic or calories from it, or it’s going to get into the large intestine and get fermented and also cause some potential negatives, but a lot of potential positives.

So why are carbohydrates important? Why are they half of what we consume? Well, they are sweeteners; food preservation -- you know, some of the discussion this morning was salt and sugar in foods. There are some advantages for sugars -- you know, think of jam, how long it can sit around because the sugar is tying it up. Functional attributes, viscosity, texture, body, browning capacity; a lot of the foods we like because of the carbohydrates have these components in them; energy, and then just this fermentation; what goes on in the large intestinal tract.

Is there a carbohydrate requirement? If you go back in nutrition and if you are as old as I am, you
can go back and say there is no technical requirement, because we can make carbohydrate from protein. So technically, you could take protein, deaminate it and use it for gluconeogenesis; but, if you go into the 2002 DRIs they thought about it some more and said, actually we know that there are parts of the body that do require carbohydrates, so let’s put some carbohydrates in, as required, so 130 grams a day, a very small amount, in the DRIs. We don’t know exactly -- if you look how much do we need, we don’t know exactly how much is necessary for optimum health, but we know -- we had a little discussion about ketones already; that carbohydrates are a good source of energy, and that if there are none around, you do become -- use ketone bodies in breaking down body fats. So, it’s worth knowing about that.

All right, glycemic index. I’m going to start on some different parts that were within the 2005 and discuss that. If you look at glycemic index, it really gets into that glycemic response. So you eat a carbohydrate and what happens? We always want to measure something. So the nice thing about the
glycemic index is something we can measure. So we bring in people that are fasted, we feed them a certain amount of the food that associates with the amount of carbohydrate and we look at glycemic response relative to a control, and a control is going to be glucose or white bread; a glucose compound that’s quickly absorbed. So you can see white bread gets a score of 1 there. So that’s our base and then we compare everything to white bread. And if you go up and down, potatoes and rice typically are the ones that are digested and absorbed more quickly, they get a higher score, which is typically considered not positive. And if you go down the line, mixed foods like cheese pizza are going to be less; sucrose is going to be less, because it has some fructose in it, and fructose alone is going to be a lot less. So glycemic index is really driven by the glucose and fructose content of that food and how quickly it’s digested.

Glycemic load really just corrects for serving size. So carrots -- whenever I give my sports nutrition talks, people say, are carrots bad because of their high glycemic index? And, no, carrots aren’t
bad, but the food, the carbohydrates in them is mostly starch glucose, so they are going to be higher than fructose. So when you actually calculate out glycemic load, you can see carrots have a low glycemic load just because the serving size is corrected for in glycemic load. And, glycemic load is really a good indication obviously of calories too, so.

So, if you go back to the definitions and the recommendations, there is in the DRIS a UL, where there was not a UL based on glycemic index; and it said because the critical mass of evidence necessary for recommending substantial dietary change is not available. There wasn’t enough information. But, they did say, principle of slowing carbohydrate absorption is potentially important, further research is needed.

This, because of the emerging science, is something to consider, types of carbohydrate or characterizing their glycemic potential is of interest. And I think we definitely see a movement towards types of carbohydrates. We know carbohydrates are a really diverse group, and how to put them into groups that are useful to get at physiological effects and help us come
up with recommendations we want to make for carbohydrates.

Glycemic response, lots of things affected, just the types of starch that are in the food intact. Large particle size will affect it, if you can’t digest it and absorb it, it’s going to slow down the process, so if it’s intact -- raw starch, you know, the effect of cooking. If you don’t cook starch, you eat a raw potato, see what it does to you. It’s not good. You don’t break it down. You don’t get any calories from it, but you get other problems. And just the interaction, the more complicated the food is, you are going to get a lower glycemic response. But there is no recommendation or UL for glycemic index or glycemic load.

So what did we get in the DRI report? We have an RDA for a carbohydrate; this acceptable macronutrient distribution range we’ll talk about; there is a recommendation for added sugar consumption, but no recommendation for an upper limit for a glycemic index or glycemic load. So, acceptable macronutrient distribution ranges, carbohydrates are most of your
1 calories, so it’s really important -- I always feel
2 like I am the lone person -- here I’ve got the market
3 share of the calories and nobody cares about me. It’s
4 really sad. So maybe I can convince people they should
care. But, the recommendation is 45 to 65 percent of
5 calories should come from carbohydrates; and then
6 lipids 20 to 35; proteins, 10 to 35. Why -- where did
7 we get to these levels? We know that below 45 percent
8 I am not going to get my adequate intake for fiber, and
9 based on our discussion yesterday, fiber continues to
10 be a problem. So, getting people to eat more
11 carbohydrates is an important part of getting them to
eat the fiber that they need, and just the -- you know,
12 like at the end of the day, we’ve got carbohydrates,
13 lipid, protein and alcohol, so take a pick. I mean,
14 where do you want to put them in? So, if carbohydrates
15 go down, lipids and protein have to go up. So, there
16 is no real reason to do that. Higher than 65, if you
17 go really high, there is some data on the high
18 triglycerides people get concerned about, and that you
19 decrease fat and protein to too low of levels.
20 Added sugars -- this is very controversial and
difficult, because this is considered sugars and syrups that are added to foods during processing or preparation; soft drinks, cakes, cookies, pies, dairy desserts, candy. You have heard from Christine that that’s a lot of what people consume and kids too. Recommendation for added sugars is that they not be more than 25 percent of total calories. And this is looking at -- this is from the DRI report -- looking at added sugar intake combined with nutrient intake data. This is being reassessed in other groups. But, if you look at this calcium intake as they get beyond 25 percent of their calories as added sugar, calcium intake goes down; so it’s based on that calculation; that if we get sugar above 25 percent of total calories, then it’s hard to get nutrients. The significance of added sugar to human health, this was from the 2005 Dietary Guidelines, so they suggested there was a positive association between the consumption of sugar-sweetened beverages and weight gain, a reduced intake of added sugar, especially sugar-sweetened beverages may be helpful in achieving recommended intake of nutrients and in weight control.
So that’s right out of the 2005, and it really was based on the study that came out, that’s quoted, or that’s one of the data points. And this is the nurse’s health study where they reported that those who increased their sugar-sweetened soft drink consumption from low to high, which was really one, less than one, greater than one, had more weight gain. So, higher consumption of sweetened, sugar-sweetened beverages, this is associated with greater weight gain and increased risk for Type 2 diabetes.

So, discretionary calories really fits into this category here. Added sugars fit into the category of discretionary calories, because they are part of the difference between a person’s energy requirement and his essential calories. Persons who are sedentary have very few discretionary calories, and I think you have heard about that already. The Energy Committee that -- but maybe this concept is too confusing and not helpful. You know, it seems pretty easy, but probably not.

So, where do we end up on dietary carbohydrates? We have an RDA, 130 grams a day; we have a range of
intakes, 45 to 65 percent; added sugar, 25 percent or less; and these are existing dietary guidelines DRI-type recommendations; dietary fibers based on K-cals, 38 grams for men; 25 for women; and pretty, you know, good support that carbohydrates really are the primary calorie source; that they are less expensive. There is no reason for us to have less than about 50 percent of our calories as carbohydrates. So then the challenge is just managing that 50 percent of our calories, how to make that the best.

So we are going to talk about fiber, of course. What is fiber? Carbohydrates and lignin that escape digestion but may get fermented in the gut. According to the 2002 DRIs, it’s a nutrient, so fiber moved up. On the nutrition facts panel, 25 grams is the daily value. A marker of a healthy diet -- overall, when we look at healthy diets and a lot of diseases they are plant-based diets, which are higher fiber diets. So fiber tends to be protective for a lot of diseases, and fiber does come along with other things. So whenever -- you know, maybe it’s just a marker. Fiber itself may not be the active component. We do have health
claims for oats, barley and psyllium, for their cholesterol lowering. I already told you that the IOM separated out dietary fiber and functional fiber and dietary fiber is non-digestible carbohydrates and lignin that are intrinsic and intact in plants. So that’s saying we want people to eat a plant-based diet; they should get their fiber from plant foods. Functional fiber can come from anything, so it’s the isolated non-digestible carbohydrates that should have a beneficial effect. It can come from plants; it can come from bacteria; it can come from yeast; total fiber is the sum of those.

Issues -- some of the fiber issues intact and naturally occurring in foods -- this makes it kind of hard to measure and put on a label. It’s more of a concept than it is something we can put a handle on better than that. Go back to the 1970s, the dietary fiber hypothesis was based on populations consuming unrefined diets that were high in fiber and slowly digested carbohydrates, so different attributes to these diets. Fiber has lots of biologically active compounds, and we know that fiber within the plant’s
cell structure is handled differently in the body than
isolated fiber, so fiber is not all alike, just like
carbohydrate is not all alike.

The recommendation for 14 was based on this data
showing protection from coronary heart disease, so
these were three large perspective studies that were
put together in this data set, and you can see as fiber
intake goes up relative risk of coronary heart disease
goes down. This is just from the -- this was from the
DRI. We have this recommendation of 14 grams of fiber
per thousand K-cals. There is no recommended UL for
total fiber, so when the DRI committee got together, we
know that there are occasional adverse GI symptoms --
that’s gastrointestinal, not glycemic index -- observed
when humans consume isolated or synthetic fibers. So,
this is a review we just published. In looking at
different fibers in high enough concentrations they
cause problems. When the DRI committee thought about
that, they said though that due to the bulky nature of
fiber in foods, excessive consumption is likely to be
self-limiting. And since fiber intake tends to be
really low anyway, I don’t think there was any concern
of getting people too high, because most people are so low to start with.

New carbohydrate information since the 2010 Dietary Guidelines. So that was the lecture. My kids always say, you know, enough. Do it at work. Keep it to yourself. So anyway, what’s different? So I have tried to pull together some papers I think that have changed the -- that have been published since the 2005 Dietary Guidelines and have -- we should consider in our deliberations. The general areas they are in are sugar, especially fructose; glycemic index/glycemic load -- I want to follow-up on that; dietary fiber, whole grains; and also food form, liquid versus solid, and I know we overlap with other committees on that.

Macronutrients And Obesity -- oh -- I actually tried to do a review on this before. It’s very difficult obviously, but there is no clear evidence -- this is a review that was published in the European Journal of Clinical Nutrition that I thought had some good points; no clear evidence that altering the proportion of total carbohydrate is an important determinant of energy intake. And, if you get into
this literature -- there are lots of studies out there -- but, you know -- like proteins versus carbohydrate, if calories are controlled, obviously it is not very clear that micromanaging your macronutrients does that much for weight loss. There is evidence that sugar-sweetened beverages do not induce satiety, and I want to talk a little more about that. There is a lot of controversy there, but quite a few studies in that area. Findings from studies on glycemic index on body weight have been inconsistent. So I think since the 2005 Dietary Guidelines that data has definitely not been very positive that that is the way to go, and dietary fiber intake is consistently linked to less weight gain, but you know, we said lots of dietary fiber diets have other positives besides just the fiber.

I had to use this slide, because it was published in our journal, and how bad is fructose? It’s like when do you stop, you know, when did you stop beating your wife? It’s the same kind of thing that, you know, we are assuming it’s bad and trying to sort this out of all the different carbohydrates, a couple of other
disagreeing viewpoints, so I would suggest you read that one by Dr. Brave. And also Dr. Anderson, in a similar version of the journal said, there is no evidence that the ratio of fructose and glucose consumed from sugars has changed over the past four decades as a result of high fructose corn syrup replacing sucrose in many applications. So trying to get at this data, and I think it would be good if we have some people come in and speak to our committee on this, is that high fructose corn sweetener is pretty much the same as sucrose, so if you are just doing a switchover there aren’t big changes in your exposure of fructose and glucose. So, high fructose corn sweeteners does not appear to contribute to overweight and obesity any differently from other energy sources in this review and critical reviews in Food, Science and Nutrition that calories are calories and high fructose corn sweeteners are no different than other calories, calories per calorie, so that was their review.

This is another -- sugar-sweetened beverages and body mass index in children and adolescents -- a meta
analysis that was recently published, they looked at all the trials they could find. There were 12 trials that were in this reference; 10 were longitudinal and two were randomized trials, of sugar-sweetened beverages and weight gain in children and adolescents. Quantitative meta analysis and qualitative review found that the association between sugar-sweetened beverages and body mass index was near zero. You know, there is not a lot of studies there, and remember too that the data on this, you know, if you think how can people come up with such a difference, calories do count, but in this the sugar-sweetened beverages were not, you know, linked to body mass index.

Obesity Review’s recent article -- they reviewed associations between intake of calorically-sweetened beverages and obesity relative to adjustment for energy intake. And this is difficult, to try to adjust for energy intake and put that in perspective. They found there were 14 prospective and five experimental studies that were reviewed in their paper. They felt like a high intake of calorically-sweetened beverages can be regarded as a determinant for obesity, but this is --
1 if you read this, this is kind of confusing -- no
2 support that the association between the intake of
3 calorically-sweetened beverages and obesity is mediated
4 via increased energy intake. They suggest there are
5 alternative biological mechanisms. So you can see
6 within the literature there is a lot of disagreement,
7 you know, basically looking at the same data.
8
9 This is a research editorial also published in my
10 journal, so how discretionary can we be with sweetened
11 beverages for children, and this is a quote directly
12 from it. Based on cumulative evidence, it is
13 recommended that children consume no more than one
14 sweetened beverage per week. There is little room, if
15 any, in the diets of children to replace healthy foods
16 with the empty calories from liquid sugar. So I don’t
17 know if -- what exactly -- is that chocolate milk? I
18 mean, what’s milk, besides milk has lactose, so is that
19 sweetened or is it only if I put other sugar into it?
20 So, I think there is a lot of -- this whole area of
21 sweetened beverages sugar intake is -- there are a lot
22 of things being published. There is a lot of -- people
23 feel very passionate about it, but for us to step back
and take a research-based view of it, I think is going
to take quite a bit of effort in looking and reviewing
what is out there.

I wanted to put this in just because I think --
the way I always think of diets is that protein is very
important. We never want to lose sight of that. And
since we don’t have a protein committee, I’m going to
be the protein person too. So, I want to point out
that diets -- protein is the most important thing when
we put a diet together, so we definitely want to make
sure we talk about that.

On low-calorie diets higher protein intakes are
recommended, so as calories go down protein goes up.
So, in saying, is it carbohydrate versus protein on
low-calorie diets, which we are going to recommend,
protein has to become more important. It’s going to be
a higher percentage of the K-cals that somebody can
consume. There are probably some advantages over
carbohydrates in satiety, you know, depending on what
types of carbohydrates are chosen, increase of
thermogenesis, maintenance of fat-free mass. So this
review I think did a good job of summarizing that there
are definite advantages to high protein diets that we don’t want to lose sight of and that, in weight loss, we want to make sure we are not breaking down body protein, because that’s going to help people burn calories.

This is a little bit on glycemic index. I wanted to start with sugars, and I guess you already know that it’s pretty controversial on sugars, but I don’t think we have data that suggest that any one sugar is the bad guy, and that if we just get rid of one sugar our lives will be better; that it’s calories and needing to give carbohydrates a better way of looking at carbohydrates, but not just picking on one.

Glycemic response in health, a systematic review is meta analysis recently published, among glycemic index studies the observed reductions in glycemic load are most often not solely due to substitution of high for low glycemic carbohydrate foods. The big thing is available carbohydrate obviously, if you don’t control for that. And also in this review they talk about fiber and unavailable carbohydrate, that that’s an important overlay to all of the glycemic index because
we know that typically fibers do lower it, but total calories, total carbohydrate the important thing.

This was a recent thing looking at glycemic index/glycemic load, and this was in the Women’s Health Initiative and what they, over about eight years of follow-up, there were 1,476 incidental cases of colorectal cancer, and they looked at total carbohydrate, glycemic index, glycemic load, intake of sugars, fiber and there was no association at all. They concluded there was no -- results do not support that diet characterized by high glycemic index or load plays a role. And this was in post-menopausal women.

Another review just published in Nutrition Reviews, a very extensive review on glycemic index and glycemic load and dietary recommendations. They looked at the epidemiological data and glycemic index, glycemic load and all the relationships, the diseases, heart disease, insulin sensitivity, Type 2 diabetes, dyslipidemia and obesity, with initially healthy people that were followed, very mixed results. The only positive association they found between glycemic index was with the development of Type 2 diabetes, that that
was consistent, but otherwise the data was not consistent, and their take home message is it seems premature to include glycemic index or glycemic load in dietary recommendations.

I want to just finish on fiber and satiety. So this is all the things that fiber does to affect satiety, and up on the top there you can see hormonal intrinsic and colonic effects. And this is what’s so complicated when we look at different types of carbohydrates with or without fiber is they have effects throughout the digestive tract that could help us in making people feel better eating less, kind of I think where we are going to end up here. The left, the hormonal effects; the middle, the intrinsic effects, just the chewing; and then on the right, the colonic effects, and I think we have ignored the colonic effects. Nobody really likes to collect poop, that’s what I say, so in nutrition that, you know, like it’s a black box and we stay away from it as much as possible, but what’s going on down there is actually pretty important.

Cereal grains of weight management; whole grains
associated with lower body mass index, waist circumference, risk of being overweight -- so there is pretty consistent fiber and whole grains preventing weight gain, helping with weight loss. This was from the Women’s Health trial, post-menopausal women at 12-month intervention, and this is this mixed diet idea that it’s not one particular thing. They are on a low-fat diet and that the dietary correlates, everything comes along for the ride. So, it’s low-fat, it’s high fiber from a mixture of whole grains, fruits and vegetables; a higher fiber intake; lower body weight; and there were some improvements in biomarkers, so.

This was a study done -- and this is difficult, because a lot of studies with whole foods are not real successful. This one was done at Penn State, Penny Kris-Etherton. They looked at obese adults and gave them a whole grain diet of refined grain. One thing, and it’s really right down there on the bottom, both diets improved CVD risk factors, and this typically happens on even refined -- you know, people think, okay, what’s the difference between whole grains and refined grains -- both groups when you put them on
these controlled diets do better. So, the whole grain diet did a little better in C-reactive protein and a little bit of difference in body fat, but overall they were both showing improvements.

And this is another study that was published since 2005 on whole grains, you know, where they actually gave people whole grains or refined grains for six weeks and saw virtually no changes at all. And, you know, these diets are not that different. You know, whole grains are important, you get more fiber. But, in these types of studies, you see improvements just when you put people on these refined grain diets that are typically better than the diets they are on.

Okay. Satiety -- I want to just finish up on that -- what makes people stop eating and feel that they are full, and it's a self report. Usually you ask people a combination of questions, hunger, satisfaction, fullness, desire to eat? I already talked a little bit about this, but these things can happen; fiber effects throughout the digestive tract. So where it's having its effect we don't know, but throughout the digestive tract we see differences. And it's usually -- this is
what it is; it’s a visual analog self-report and you compare different treatments. Because, you know, I always get this question, well aren’t people always hungry or always full? Yeah, you know, there are differences among people, but if you use the same person in these trials and they come in fasted, they are given the treatment, it’s how to -- what’s the acute effect of that treatment. I go back really -- even though I know I am supposed to stay within 2005, I really like old stuff. I am more of a history buff than anything, and this is a poorly, you know 10 subjects, but I think it’s kind of a neat little study where they compared apples, apple puree and apple juice and they found that -- you know and it was 60 grams of available carbohydrate, the juice could be consumed 11 times faster than the intact apples and four times faster than the puree. So just kind of, you know, what makes people slow down and not eat so much, you know, fiber and whole foods. And when they actually controlled the rate of ingestion so that it was all equalized, the juice was less satisfying than the puree and that was, you know, less than the apples.
There is a more recent study in carrots that kind of gets at this physical structure of real food, and in this they were given either carrots at 200 grams or whole carrots, blended carrots or carrot nutrients. So carrot nutrient is like a carrot cocktail. You figure out all the nutrients that are in it, throw it in a drink and it’s a carrot cocktail. What they found, whole carrots and blended carrots resulted in significantly higher satiety, and when you look at food intake throughout the rest of the day, the carrot nutrients didn’t really affect it at all, so it was the fiber content and the structure of the food that were important.

This is another recent one on whole grains that I wanted to bring in, and this is another thing where they controlled different types of breads. So it was white bread, whole meal, wheat bread and then whole kernel bread, which is like bread with chunks in it, and what they found is the whole kernel bread resulted in significantly higher satiety than the whole meal, wheat bread or white bread, so actually having some structure into the food. And they saw no differences
in blood glucose, so it wasn’t related to the blood glucose response.

I’m going to end up a little bit on viscosity just because we know that these viscous fibers do help enhance satiety, so that kind of gives some structure within the digestive tract. If you take a thing like guar gum that’s very viscous and you hydrolyze it, it’s not as satiating, even though the fiber content doesn’t change. So the fiber is the same, but when you modify it, it doesn’t have the same effect on satiety. And, if we look at gastric emptying, that’s not the whole explanation, because there are studies where they have control for that and it doesn’t explain it all.

A little bit on protein and fiber -- if you look at satiety besides fiber, there is quite a bit of data on protein, and this lupin-enriched bread that was done, it’s higher in both fiber and protein, so we don’t know which it is that’s having the effect, but this lupin bread it was higher satiety and after -- you know, you look at energy intake at lunch, they ate less after they had the lupin bread, and there were some changes in gut hormones also with grellin (ph) in that
lupin.

Does dose matter? Absolutely. You know, little bits of fiber really don’t make a difference. Here is 4.5 guar, which is a very effective fiber in a breakfast bar, but no differences. Usually it’s high doses. When we look at the DRI for fiber and we think, wow, 38 grams is a lot of fiber, but to really increase fullness you see higher doses over the day being more successful than lower doses.

And these are the ones that have been shown. We did a review trying to look at all the fibers. The viscous fibers for sure worked. Wheat brain definitely works. The pea fiber, which is more of an insoluble fiber; cellulose, soy polysaccharide have some data, but it’s generally in higher doses.

I’m going to finish up on fiber intake in the U.S. is low. It’s about 15 grams per day; recommended intakes, 25 to 35. Most fiber-containing foods are pretty low; one to three grams of fiber. USDA data shows that white flour and white potatoes provide the most fiber in the U.S. diet, not because they are concentrated fiber sources, but because they are widely
consumed. So we haven’t made a lot of progress in getting people to eat high fiber foods. So any push towards that -- and because of that there is interest in the addition of functional fibers; if we can’t change people’s food intake, let’s change their food. So -- and this is all reviewed in this Health Implication of Dietary Fibers paper.

A little bit on just -- I want to finish up on satiety -- just I stuck these in here since I am talking about satiety. In this research they compared beverages with sucrose or high fructose corn sweeteners on hunger, satiety and energy intake; no differences between sucrose and high fructose corn sweeteners, which really, if you think about it, how, why would you have? I mean, it’s chemically the same, so not too surprising. Diet cola and no beverage -- if they weren’t given calories they ate more at lunch, which also seems pretty obvious, but you know, the calorie dose they got, whether it was from corn sweeteners or sucrose, no differences, but they ate less at lunch with that.

And this was kind of interesting that was just
published, because they compared high fructose corn
sweeteners, sucrose and milk pre-loads. In this study
they added a milk -- and these are isoenergetic drinks,
so they controlled calories and they saw no differences
on changes for gut hormones between these different
drinks. So, you know, looking at all these differences
there were no differences at all in satiety and gut
hormones.

So, just to finish up, high carbohydrate diets are
recommended by the DRIs. I don’t -- I think that’s --
we really need to support that. It makes sense overall
to be up in that range. Dietary fiber intakes are less
than half of recommended levels, so getting progress on
that is important. Some of the definitions from
before, from the 2005, I definitely want to include the
legumes. They didn’t get listed, but I think they need
to be up there as a good fiber source. And just
choosing carbohydrates wisely; getting people to take
the higher fiber ones. And I think that there is every
reason to push towards whole grains as a grain category
just because there is more fiber, there are more
nutrients in that category. Measures of carbohydrate
quality remain elusive, so trying to give carbohydrates 
grades is really tough and people feel strongly about 
it, but I don’t think glycemic index or glycemic load 
are going to help us categorize. Thank you. And I 
have other people on my committee, other comments, I 
welcome.

DR. VAN HORN: Great job. That was absolutely 
wonderful. Other people on the committee that either 
were on the subcommittee. Rafael?

DR. PEREZ-ESCAMILLA: Joanne, you presented data 
regarding the consumption of sweetened beverages by 
children and relationship or lack of a relationship 
with body mass index, but what about insulin resistance 
Type 2 diabetes in childhood; have you examined that 
data?

DR. SLAVIN: Yeah. Go ahead. Yeah, the question 
had to do with is there data in children with 
resistance and consumption of sweetened beverages. I 
think it’s probably in those reviews if there was any 
data out there, but I don’t have it off the top of my 
head.

DR. PI-SUNYER: There is certainly no longitudinal
data. There is some association data across the spectrum that is determinately good. So I think the evidence is pretty weak mostly because there is -- there haven’t been enough studies that have been either interventional or observational among children.

DR. VAN HORN: Certainly the diet data suggests that children are equally poor in meeting the, you know, fiber goals as the adults, and they too don’t eat enough of it.

DR. PI-SUNYER: I wanted to comment -- this is Xavier -- I wanted to comment on Joanne’s mention about the importance of protein, but you know, I think the mix-up of the message to tell people, you know, lower your calories, but take more protein, I’m not sure is a reasonable message. First of all, Americans are eating more protein by far than they need, on the whole. And I think if you give an across-the-board recommendation, it’s usually easier than trying to specify specific groups or macronutrients. So, I’m not sure that we would be doing anybody a disfavor if we said, you know, across the board cut back your portion sizes if you have an appropriate proportion of fat, carbohydrate and
protein and not try to push protein a little bit, particularly since with protein very often comes fat and saturated fat.

DR. SLAVIN: I just -- I wanted to comment on Christine’s with the milk consumption though, because I think the foods that we choose are really a problem, and if kids were consuming milk, that’s a protein source. But, as you look at her data, as they have switched out of that, protein needs in general, the average is good, but there are people that just aren’t getting enough protein and a lot of it has to do with bad food choices. So, I think, as the Dietary Guidelines group, we have to keep that kind of front and center; that as people cut calories, you know, we don’t really want them -- and that’s kind of why I like the discretionary calories idea that we want them to cut calories that aren’t essential nutrients, which protein is.

DR. VAN HORN: One thing that I am aware of that perhaps we need to bring the data up to speed since the 2005 Guidelines is the importance of plant-based protein, and the data -- certainly the data that I am
I aware of, both with Cardia and Eric, I think, and Intermap definitely illustrating that higher -- that plant-based protein is interestingly associated with both blood pressure, cardiovascular disease and obesity. So, you know, it would appear that the message of more plant-based foods transcends not only the message of, you know, improving carbohydrate intake, et cetera, but also by preferentially increasing protein from plant-based sources, there is a benefit as well. Yes.

DR. NELSON: I agree with that. Thank you so much. This is Mim Nelson. So, I think that somehow we do -- I guess I’m trying to put a point on some stuff you said, but we have to figure out a different way to frame carbohydrates, because I think that this message that, you know, we need to have a diet that’s rich in carbohydrates hasn’t worked. And, it’s because when you think about it, I mean, the carbohydrates we are trying to get them to eat; fruits and vegetables, low and, you know, non-fat dairy, whole grains, legumes, you know, all the sort of more nutrient-dense carbohydrates, we obviously aren’t getting there. And
it’s the abundance of high-calorie caloric -- whether it’s sugar; whether it’s just refined grains; whether it’s, you know, snack foods, everything else, all of these other carbohydrates that really would go into -- you know, white bread, white pasta, you name it, sort of that goes into the discretionary carbohydrates. If we do just talk about carbohydrates, in a sense, there is -- the balance is just so off from what the American population is eating that I think we do have to be really careful about how we present them or look at the evidence, and you know, really around those food groups that are so important for health. And the other, you know, just look at the grocery store and the way our food supply has changed. It’s really the preponderances in these snack foods and refined everything else and that’s -- I think it’s a real problem in energy intake. We just eat a lot more of those foods and not enough of the good ones. So I think we have to be very careful on how we frame carbohydrates, as opposed to just carbohydrates, it’s more these food groups and not these.

DR. SLAVIN: Yeah. And we were talking on the way
over just about complex carbohydrates, you know, like how do you make the point? And glycemic index is good in some ways, because it does -- you know, it doesn’t solve the problem, but it does tend to get you more there. But I agree that, you know, with -- and that’s why I guess I put in this idea about proteins is that a lot of times we want people to reduce calories, but really not across the board; that as you reduce calories, protein -- the percentage has to go up. So -- especially for kids. I just think -- and you know, I think that the vegetable protein data is interesting, but protein quality, you know, if you are going to limit protein and limit calories, then protein quality becomes a more important data point that we have to consider, especially for children, for kids, pregnancy.

DR. NELSON: But I would say equally as important is carbohydrate quality.

DR. SLAVIN: Yes, yes, for sure.

DR. VAN HORN: Cheryl.

DR. ACHTERBERG: Thank you. Cheryl Achterberg. I wanted to reinforce what Mim said, and I think maybe we should be brave enough to consider looking at starchy
food components and consider putting potatoes and bread together, at least look at that as an option, since most of the world does already.

Two questions really more than anything else. I know, Joanne, you presented a very very comprehensive review, but I also know that in Europe they seem to be a little less squeamish about doing gut health-type studies and that I think the Committee needs to take a good look at what some of those European studies are saying about fiber. I just think they have a lot more data that’s come out recently, but I am not sure that Americans are that aware of it yet.

I also want to ask the question, if you are aware of any studies so far -- I noticed on one slide you mentioned resistant starch -- whether any studies have been completed yet that look at resistant starch in the context of a whole diet, as opposed to a meal or a food, and what we might make out of that?

DR. SLAVIN: Well, you know, I think that gets into this resistant -- that starch complex carbohydrate, you know, what’s good about complex carbohydrate that it’s got resistant starch in it. So
there are some data on intakes. Most of the biological
data is short-term, but you know, if you look at people
that have high resistant starch diets, there are
people, kind of like the 1970s fiber hypothesis;
unrefined plant-based foods are going to be high in
fiber and resistant starch. There are method issues
that have limited just databases on, you know, intakes
of resistant starch, but it definitely goes along with
high fiber and more plant-based diets.

      DR. ACHTERBERG: And there are some people trying
to tie it to perhaps preventing Type 2 diabetes?
      DR. SLAVIN: Yeah. You know, I think that the
whole -- I think with all the information we have
that’s a number really important with our group to
consider. The carbohydrate message, trying to separate
it from over -- you know, because we are most of the
calories that people eat, so within -- if we limit
calories and control calories, which carbohydrates
would be the most protective, and it, you know,
obviously fiber for sure and resistant starch, which
isn’t going to affect glycemic response. I mean, if
it’s a truly resistant starch it goes straight through
and it doesn’t change insulin; it doesn’t change blood glucose, so it would be a positive for sure.

DR. VAN HORN: Roger?

DR. CLEMENS: Joanne -- Rog -- thank you very much for that excellent presentation. I appreciate the comments that, in fact, not all carbohydrates -- some carbohydrates function as preservatives. They actually lower the AW and so prevent microbial growth. And I appreciate your comments about the betaglucans and the structural differences there. As we all know, not all betaglucans are the same. Some are immunomodulatory from different sources; others may affect dyslipidemia, as we are all concerned about.

Also, some amino saccharides have been demonstrated to inhibit dental caries and actually inhibit the adhesion of strepmutans through the dentition, something we might want to consider or at least mention in our comments.

And lastly, I would like to comment, as you pointed out so nicely about the shortchanged fatty acids, and as we know, the micro flora in the G.I. tract has a tremendous impact on gut health and overall...
health, and that too may be a common area our group wishes to address then as we look at more and like impact of whole grains in affecting our microbial health in the G.I. tract.

DR. SLAVIN: I appreciate that. Thank you.

DR. VAN HORN: Chris?

DR. WILLIAMS: Christine Williams. I wanted to comment on dietary fiber intake in children, which of course is very low compared with the National Academy of Science Guidelines of 14 grams per 1,000 calories. On the other hand, there isn’t a whole lot of scientific evidence that those guidelines are appropriate, especially for young children, which might be one thing we might want to look at. Dietary fiber certainly has the same health benefits in children as in adults and we need to encourage higher levels.

DR. SLAVIN: Yeah. I appreciate that, Christine, because the -- you know, since those recommendations were made on 14 grams per thousand K-cals, it was just a math, so you know, it was never really thought about for kids. So the ones for little kids are way too high, and I think the old recommendations of age plus
five actually make a lot more sense than the ones based on K-cals.

DR. NELSON: Right.

DR. VAN HORN: Larry?

DR. APPEL: Yeah -- Larry Appel. I was just curious about this review in which you said that the only consistent association of G.I. was with incident Type 2 diabetes, which you know, I had thought was -- I thought that it was pretty inconsistent results. So, I mean, is this independent of fiber volume mass index? I mean, is this something we are going to act upon? Because I was -- I mean, obviously Type 2 diabetes is important. It’s on the front page of the newspaper today.

DR. SLAVIN: Yes. No. I saw that, and I -- you know, I think we need to consider whether, like in that review, the fiber and other things were taken out. Because I think, you know, glycemic index, if it can help us we should absolutely use it. But I think overall, especially on weight control, that it -- you know, because the problem you have in an intervention study, which people do, is the easiest way to get GI
down is to put fructose in the diet. So that’s, you know, a lot of the intervention studies that are done are really not consistent with the epidemiology, so. But, you know -- yes?

DR. NELSON: Just a caution about fiber, and I’m not a fiber expert, but I -- you know, one can’t help but look at what’s happening in the food supply. You know fiber is now being added to, you know, artificial sweeteners, and it’s being added to everything, and while I think that the data is so strong around fiber and health that I want to make sure that whatever we consider that we are thinking more of it as part of our food supply as opposed to as an isolated additive to the food.

DR. SLAVIN: And, you know, I think we can use the DRI Committee for that too; that they are saying that not all fiber is the same; the dietary fiber in foods, push that, push that, there is no real disagreement on that. The functional fibers, unless they are shown to do something, we shouldn’t just accept them as good; that they actually need to have a physiology effect before we green light them.
DR. VAN HORN: One more question and then we’ll take a break.

DR. RIMM: Go back to the -- this is Eric Rimm -- go back to the issue that Larry brought up about GI and diabetes. I think one of the issues with glycemic index and one of the challenges of using it epidemiologically or otherwise is that it’s probably much more important to people who are insulin-resistant. So, if you look at the glycemic index of somebody who doesn’t exercise or has a BMI of 29, it’s probably a much more, a much stronger predictor of diabetes and other outcomes than it is among someone who is a marathon runner or does strength training, or has a BMI of 22. So, it really is sort of an effect modification by where you are at; and given that 60 percent of the population is now overweight it is maybe important enough at the population level.

DR. VAN HORN: All right. Well that was absolutely wonderful. Thank you very much, Joanne, and everyone for your input. We are scheduled to take a break. We had a little one before, so let’s keep this to about ten minutes, and then we’ll start back on
fatty acids. Thank you.
(Whereupon, at 10:53 a.m., a brief recess is taken).

DR. VAN HORN: All right. Thank you all. We are going to get started now with Dr. Pearson leading a discussion about fatty acids, along with Drs. Rimm and Clemens.

DR. PEARSON: Thank you, and on behalf of my subcommittee, Eric Rimm and Roger Clemens, we are going to have a little different --

DR. VAN HORN: They can’t hear in the audience.

DR. PEARSON: Okay. Thank you. On behalf of our fat subcommittee, which is Roger Clemens and Eric Rimm, we are going to have a little bit of a different format to where they facilitate a discussion. I am going to stand up here so I have my pointer. We could go through this quite large and complex issue of fats.

But just to start out with maybe a little bit of a comment, and that is, a year or so ago -- actually, for the two years before that -- we had a project in which we sought out a low incidence coronary population to really examine how one could in fact prevent the onset
of a cardiovascular epidemic and ended up with a population-based study on the country of Grenada, and basically spent three years looking for a heart attack. We failed. We talked to the physicians. We talked to the nurses. We did a 2,100 person survey, et cetera. We didn’t talk to the cardiologist, because there was none in the country. There were no cardiac surgeons. This is a country without a cardiovascular epidemic. And so, really the question one leads to as hypothesis is that there is something that has caused that. This is a country with a reasonable amount of obesity, certainly a lot of diabetes, a lot of hypertension from their Afro-Caribbean genetic basis perhaps, but no coronary disease. So, the hypothesis you are left with says the dietary consumption of saturated fat and cholesterol is really the key determinant for raising serum LDL cholesterol levels about the threshold necessary to induce atherosclerosis on a population basis, and this, for the U.S. Dietary Guidelines, leads to the question of what should that threshold be and what are we going to do nutritionally to get to that point, where our leading causes of death, which is
atherosclerosis, coronary disease and stroke are able
to, on a population basis, be controlled? So what you
are left with here then are many examples from a
population basis, in which you have excess caloric
intake, decreased caloric expenditure, high sodium
intake, perhaps heavy alcohol, with hypertension and
diabetes, also populations with a lot of smoking, but
in fact, if there is not the saturated fat and
cholesterol getting the LDL above a certain threshold,
you do not proceed into an atherosclerosis situation.
And this is not just the country of Grenada, but of
course, the countries in East Asia, Africa, et cetera,
in which you have very high levels of hypertension in
smoking, et cetera, but unless you have an LDL
cholesterol of a certain level, you are really not
developing a coronary epidemic.

So I think the real question is, is that can we
develop and look at in somewhat of a controversial
issue the dietary patterns in which to lower the
cholesterol, the total cholesterol in the United
States, to about the levels of Japan, et cetera, 160
milligrams per deciliter, so -- through the Dietary
Guidelines of saturated fat and try to get below that threshold, where we can start to see big changes in the levels of atherosclerotic cardiovascular disease.

So what we are going to do with our facilitative discussion here is talk about dietary lipids; and you can see that this is the splitter’s view of dietary fats, similar to what Joanne showed with fiber, which is just a huge number of compounds; try to go through this in a systematic way in identifying the various components we might want to work with. Now, just one point is that there are a couple of instances with the whole area of fats, in which this has gone into the pharmacologic realm. This particularly deals with plant sterols and stanols and omega-3 fatty acids, which are oftentimes given in pharmacologic doses, and we are really not going to talk about those. This is really a -- it’s really almost a pharmacologic issue.

So let’s maybe look at this relatively complex view of dietary fats and maybe focus over here on the sterols, and just make a comment on the plant sterols. There has been some discussion on sterols and stanols, particularly in pharmacologic levels that there would
be the prevalence of some of the -- of the polymorphisms, which allow increased absorption of plant stanols and sterols, but I think this is really too low of a prevalence really to be a public health issue, and obviously we are all going to be emphasizing whole foods, fruits and vegetables for that. So, I think the plant stanols/sterols really is a discussion probably limited to some further discussions in relatively unusual genetic subgroups.

The animal sterols, of course -- the plant sterols obviously are another issue, and the current recommendations for dietary cholesterol of course is 300 milligrams a day, in general, for the population and 200 milligrams a day, if you are interested in lowering your LDL cholesterol, and I guess one of the questions is should we look at that again given some of these population issues relative to the level of dietary cholesterols we have. This was reviewed in the 2005 a bit, but there may be, with low cholesterol, dietary cholesterol diets, some opportunities to reduce that further and maybe ask Roger and Eric if you have any comments on the dietary cholesterol issue.
DR. RIMM: Yeah. This is Eric Rimm. I guess the biggest issue with all the dietary lipids, that the biggest -- there we go -- this is Eric Rimm -- I think the biggest issue with all of lipids, and maybe this will come more into play with fatty acids and other fats more than sterols, is what can we do differently; what science has been done in the last five years that we can really advance what’s already been there? And it may be that for some of the plant sterols that there is a fair bit of evidence in the last five years that we may want to change or may want to add some additional information. For cholesterol, I’m not sure if there has been a substantial amount of new literature that’s going to change the 300 milligram or 200 milligram recommendation, but you know, I guess that will -- we’ll have time over the next year to really search that out, to see if it’s worth trying to make a difference, and maybe there will be differences for kids versus adults, but I think there is a lot more new evidence in the fatty acids than the other fats area than there is for cholesterol.

DR. PEARSON: Roger?
DR. CLEMENS: Likewise that when we pick up --
Linda had mentioned earlier about plant protein,
obviously plant proteins don’t carry cholesterol, so
that may be another implication for us to look at plant
sterols that have implications on fatty acid profiles,
as well as cholesterol.

DR. PEARSON: And just to point out, the
guidelines are, of course, is that the 300 and 200 are
maximum amounts, and there certainly, I don’t think, is
any evidence for there being any basement effects.
Unlike some of the fatty acids, there really isn’t a
lower danger point in dietary cholesterol.

I think there are some other issues with other
sterols that are going to be covered I think in the
vitamin D and then with other fats in foods, the
retinols and some other fat-soluble vitamins, but I
think those will be covered by other parts of the
working groups.

I think where the issues come up a little bit more
has to do with the fatty acids. The current
recommendation is 20 to 35 percent of calories from
fats. Below 20 you do get into some of the issues of
essential fatty acid deficiencies and some clinical syndromes related to that above; also have the opportunity to have really excess carbohydrates, as Dr. Pi-Sunyer had mentioned, a high carbohydrate diet is if you don’t have the calories from fat. At the upper end, you get into the risk of very high fat diets of hypertriglyceridemia, et cetera. But obviously this is a situation in which the type of fatty acids have been particularly important in that it may not be that the, just the total fats -- percent of calories is the issue, but rather, their make-up between saturated, monounsaturated and polyunsaturated, and wanted to talk a little bit about the saturated fatty acids.

We have talked about the short chain fatty acids a little bit already. We talked about it relative to colonocyte health and colonocyte differentiations, et cetera. There is also some evidence that short chain fatty acids may modulate into HMG CoA-reductase activity at the liver level, so there may be a variety of issues, but I think these are largely carbohydrate-oriented rather than dietary fat-oriented.

We have the cholesterogenic fatty acids, and with
these -- with the C-12 or C-16, obviously there is a large literature on them being cholesterogenic, and I would imagine that there is not a lot more that we need to say about those relative to their abilities to raise LDL. As pointed out in the 2005 Guidelines, stearic acid, C-18, is an interesting fatty acid, which is currently included with the saturated fats, but has metabolically distinct activities and does not have the LDL raising effect. There is one paper cited in terms of a possibly pro-thrombotic effect, and perhaps we should -- one recommendation may be to look at this issue again relative to its inclusion with the cholesterol-raising of fatty acids or thrombogenic fatty acids, and talk about its inclusion as one of the percent of fatty acids that we’d like to reduce or not reduce relative to that percent of calories from saturated fat. So I think there is an issue out there. Any other comments from Eric or Roger on this issue?

DR. RIMM: Yeah. Well, I think -- I think -- this is Eric Rimm -- I think this is an important issue where there will be a fair bit of new evidence to look
through. You know, it’s funny that Joanne mentioned that she feels like the poor relation, because people get -- they just talk about calories with fat and protein and sort everything else falls into her bucket at the end even though that she has most of the calories, but because of that we are almost overstudied. People really have, you know, teased apart every possible calorie that you can get from fat and look at where it’s from. So -- and I think a lot of the 2005 technical report did focus on LDL. There was some discussion of LDL and HDL, but I think we can’t forget that while LDL is quite predictive, if specific saturated fatty acids also increase HDL that should be really a very important consideration, because overall, predicting cardiovascular disease it’s much stronger if you have a ratio where you have some combination of those markers, as opposed to just LDL. So, looking across the board I think that will be the case, not just in saturated fats, but to specific types of mono fats and polys and their impact on LDL and HDL is quite important.

DR. CLEMENS: The interest at that time as well,
we have seen -- this is Rog -- we also have seen a
number of papers out there relative to the
polymorphisms of synthesis and degradation of LDL and
the lipoprotein fractions, and those areas may be
considered as well.

DR. PEARSON: So the current recommendations for
saturated fat are less than 10 percent for a standard,
with less than seven percent in a situation of
therapeutic reduction in LDL. It would appear that
both in children and adults we are currently at around
12 percent, and so there is certainly some
implementation issues that we not need to, to get to
relative to reducing those.

Let’s move over to the unsaturated fats, and in
the monosaturated fats, of course we have the cis
versus the trans. There was quite a bit of attention
given to the trans fatty acids in the 2005. I would
imagine that there has been some additional studies in
this regard. Obviously there is the synthetic versus
the natural trans fatty acids, which may be a nuance to
look into, but obviously the ability of trans fatty
acids to not only raise LDL but lower HDL has been an
issue. There has also been related some morbidity mortality studies, and so the question really is, is that, you know, have all the nails been put into the coffin of trans fatty acids, or is there some additional activity, research activities that we should know about? The current recommendation is less than one percent of calories from trans, and so maybe some discussion about what further we should do in this area. Eric?

DR. RIMM: Yeah. I think -- this is Eric Rimm -- I think we have a great opportunity to give a very focused direct message on trans, because there still are lots of -- I think there still is a bit of confusion. There are only a few cities that have banned it and, you know, some sort of discussion on a local level of getting rid of partially hydrogenated oils, and I think that there is more evidence now. There are animal studies suggesting that giving, you know, an animal isocaloric diet, but one that’s higher in trans over the course of four or five years led to weight gain in monkeys. So, I think there is something about trans and getting trans in the cell membranes
that does impact metabolically some very important functions. And, if our focus is going to be on weight gain, I think that we could give a very focused message on trans so that people can look at the 2010 Dietary Guidelines and say, look, this is an important message, because I don’t think the, at least at this point, I haven’t heard that the IOM is planning on reconvening the macronutrient panel, so at this point, you know, I think that our message may be that, you know, the more focused message as opposed to the 2002, 2002 IOM report.

DR. PEARSON: Roger, and then Larry.

DR. CLEMENS: I think some additional information, if you look at the national current trends of fatty acids that should be discussed at this time, and food is global. We have to look at the implications that’s going around and what it will impact on the diet drink intake here, in the United States.

DR. PEARSON: Larry?

DR. APPEL: Yeah. A view from the 2005 Guidelines. You know, in the Blue Report, I think when we actually recommended one percent, but that got -- it
didn’t get translated into the other reports, and so if there is additional evidence that supports this, then it might actually lead to the downstream effects that we want.

The second thing, and I wanted to just follow-up on something that Eric said, and I think this is relevant to the committee, is what are the surrogate outcomes that we are going to use for decision-making? And you, Tom, said LDL cholesterol. I think that we didn’t really have as formal a discussion in 2005 as we should have about whether we would use HDL as a surrogate outcome for decision-making, and I think that also applies to triglycerides, and that we should really make the decision upstream, and then, you know, do the evidence downstream, but we have to make that call.

DR. PEARSON: That’s an excellent point. Mim?

DR. NELSON: This may be a little off point, but I think it’s important, because I think one question to ask is, there is so much awareness about trans fats now in the public, and we have several cities that are banning it, but what’s happening with the food supply
is so many manufacturers have just substituted palm kernel oil for trans fat, which is basically just as unhealthy, and there has been -- I mean, close -- I mean, it’s not terribly healthy, so -- but there has also been a fairly large environmental impact to the palm kernel oil growing, so I don’t know. We talked a little bit earlier about some environmental, you know, issues, but this is one I think in which, you know, there is fairly large ramifications for sustainability and things like that.

DR. CLEMENS: At this time, I thank you for that comment then, Mim. We have also seen a great deal in the last five years changes in the kinds of fatty acids, in addition to the palm kernel that you just mentioned. We know a number of companies actually have developed the 18-1 and related compounds so that they are doing a lot of substitutions. As a result, we have a seen a marked reduction in the trans acids incorporated into different foods stuffs.

DR. PEARSON: Naomi?

DR. FUKAGAWA: Yes. Naomi Fukagawa. I also wanted to bring up the point about trans fats though,
that we must remember that natural products are also a
source of important trans fatty acids, and in some ways
we shouldn’t send everything out, you know, don’t -- to
say that it’s all bad, because they also are an
important source, you know, from our dairy products
and --

DR. APPEL: In dairy, fish, poultry --

DR. FUKAGAWA: Yes, dairy, fish, poultry. Right.

Right. So we have to have that balance.

DR. PEARSON: Just to comment on Larry’s point, in
terms of the end points, I think, and Eric had alluded
to this, is that the LDL cholesterol was quite a
primary focus of the 2005 Guidelines. There is a
movement to redo the, from NHLBI, to do, redo the
National Cholesterol Adult Treatment Panel Guidelines,
and so those would be probably coming out in a year or
two, possibly within the range here, but certainly the
coordination of these Dietary Guidelines to, and to
adequately support those would be a good thing to do so
that those would be consistent. So, I can’t really
comment or even speculate about where HDL and
triglycerides were, but they certainly are going to be
there somewhere. So I think, Larry, your point is well taken, even though the interpretation of some of those other end points is probably a little more complicated, at least the HDL is, in terms of what an HDL cholesterol level means, but I think it’s a good point for discussion.

Let’s go on to the cis monounsaturated fats, such as the, those have been, particularly with the adult treatment panel, three guidelines have been the winner of the recommendation in terms of going up a little bit with the total fat to, up to 35 percent, but not obviously doing that with saturated fats, but rather with monounsaturated fats, and I think there is obviously a lot of metabolic data relative to the protection of your HDL and triglycerides through the increase of monos versus the increase in carbohydrates. But I don’t know of any particular advances other than showing those in many, many feeding studies. Eric or Roger, any comments on the monounsaturated fats?

DR. CLEMENS: I think the only interesting one that came out just about a month or so ago, Tom, dealing with some cis fatty acids relative to satiety,
and it might be interesting to discuss as a group.

DR. RIMM: Yeah. I think, Rog mentioned that, in an effort to get rid of trans fatty acids, the industry has changed and can’t come up with new seed and new ability to make vegetable oils that are high monos, since a lot of that is cis. So I think that it’s in the food supply and there is a fair bit of literature on it, so I think it is worth looking at the impact of high monos, not only on HDL and LDL, but on clinical end points. Now we are focusing mostly on coronary heart disease because I think that’s where the literature is, but I think it would be worthwhile to try to summarize that, because I think the literature is getting stronger on the benefits of high mono diets.

DR. PEARSON: Let’s move over to polyunsaturates and maybe talk about omega-3 fatty acids first. That literature I think has been in a couple of areas; one on epidemiologic studies treating, relating to dietary patterns with high fish consumption; and the other with relatively pharmacologic doses of fatty acids in randomized trials of clinical subgroups with some I think very encouraging findings relative to efficacy;
things like heart failure recently published in the Lancet and a variety of issues. I think these pharmacologic trials also though provide some opportunities for looking at safety and other issues, other related things at higher levels that we would probably see at the population level, so maybe in a tangential way be informative. But, Roger, I know you have had a lot of interest in this area. Any other comments on the omega-3s?

DR. CLEMENS: I think part of the education -- this is Rog Clemens -- part of the education on omega-3s may hinge as well on omega-9s and omega-6s, as well as their relationship. It is interesting to see that we might want to address enviro burden. I know that the 2005 Guidelines addressed the interconversion. We talk about from flax seed oil to alpha linolenic acid, for example, all the way down the pathway that the bioconversion in a number of population groups is somewhat compromised, that could be quite an impact. Then we go all the way down to omega-3s and the DHA at the end of the metabolic pathway. We don’t all get there at the same time, and that may be a point of
discussion for our group as well. We certainly see that while the focus of the 2005 Guidelines was on cardiovascular disease and cardiovascular health, you certainly see in the last five years a merging of data that look beyond that particular area of health and one of those areas, such as macular degeneration, as well as dementia. So I think we want to broaden our focus, as well as to broaden our particular end points that Larry mentioned.

Most recently there was -- I think within the last year there was a paper by Bill Landis, if I recall correctly, to address the omega-6, omega-3 fatty acid profile, as well as the ratio relative to the incidence of cardiovascular disease and survivability. That may be a point of conversation for our group as well.

DR. PEARSON: Eric?

DR. RIMM: Yeah, I agree. I think the evidence on omega-3s from DHA and EPA has gotten even stronger. We have done a meta analysis on that and I think that there is no question the importance of that on reducing overall mortality, as well as sudden death. You know, the issue of the omega-6 to omega-3 ratio to me may be
a little more troubling just because I hate for people
to reduce the amount of omega-6 in their diets. I
think people who have sufficient amounts of omega-3s,
the ratio actually is not that important; that there is
sort of a hypothesis about the potential increases in
oxidation, but most of the data suggests that the best
inflammatory profile is when you have high omega-3 and
high omega-6. So, I think it would be worth reviewing
that data, especially the human data, because that’s
where you see there is the greatest benefit and the
ratio is actually not that important. I think it can
become important if people have very, very low levels
of both EPA, DHA, as well as alpha linolenic, so I
think there is a great opportunity from new research in
the last five years.

DR. PEARSON: Indeed. Other questions on omega-3s
from the panel?

DR. APPEL: Yeah. Just a -- will your group
actually deal with fish, because I think that’s
actually we -- we got in a vicious circle actually in
2005 trying to figure out, you know, fish
recommendations, so.
DR. RIMM: We have two experts. We’ll take on fish.

DR. CLEMENS: Yeah. We’ll definitely take on fish.

DR. PEARSON: I’ve got a few more slides for you too. The fish will swim in.

DR. VAN HORN: Well, and also, the issue related to the confusion I think out there about plant-based versus fish-based sources in omega-3 and that the, you know, the total amount needed to, you know, be comparable is extreme.

DR. CLEMENS: Yes, it is.

DR. VAN HORN: So, I don’t think the public understands that.

DR. PEARSON: Eric.

DR. RIMM: Eric Rimm -- I wanted to make a radical point, one for which I’ll probably get kicked off the stage, but the whole issue of total fat and the 20 to 35 percent of calories from fat is one that has troubled, I guess has troubled me, because partly because I sat on that 2002 IOM Committee where we tried to come up with a range for fat, and ultimately we
decided there is not one point which is the healthiest point, which is why we came up with the range concept. But the high end, you know, why we set 35 percent of calories from fat, actually was not really based on much science; it’s based on the fact that we don’t have a lot of -- at the time we didn’t have a lot of science beyond 35 percent, and there was a concern that higher fat diets would lead to obesity. I think if you look at the science, there is actually no good human data to suggest that higher fat diets lead to obesity. If anything, higher fat diets, at 35 to 40 percent, lead to lower triglycerides because it’s a lower carbohydrate intake. So, I think we should -- I am not saying that at this point we should just say everybody eat as much fat as we want, but I think there is the dogma and that low-fat diets are beneficial, and you can go in the grocery store and see a lot of low-fat foods that are essentially just put in with high carbohydrate, highly processed sugars. So my concern is that we, over the last 30 years, have created the dogma that all fat is bad, and I think that that high end of 35 percent of calories from fat is artificial.
And, if you look at some new data that has come out from dietary patterns among people in Greece or European countries, in fact they don’t have higher rates of heart disease, yet they have healthy fats. So I think we have identified, you know, on your right side of your dietary lipids some fats which can be beneficial and which we can -- industry has figured how to put in our diets in the U.S. that, in fact, if we have good fats there is no reason to think that we need to necessarily have that high end set at 35 percent. So, I hope all of us can at least look at the science before we come up with that sort of artificial end point.

DR. PEARSON: Thank you. Let’s move on to the omega-6 fatty acids. Obviously the recommendation is less than 10 percent of calories from that, mostly on the basis of some concerns about, at least relative to monos, the lowering of HDL and some data relative to increased tumor production in high omega-6 groups. Any other comments from the panel on that, Eric or Roger?

DR. FUKAGAWA: What are your -- this is Naomi.

DR. PEARSON: Naomi.
DR. FUKAGAWA: -- your thoughts about the fortification of infant formula? Is that something --

DR. CLEMENS: This is Rog. That’s a group that wasn’t within our age review. I certainly could address it, but it’s not within our charge.

DR. APPEL: Unless they are still drinking formula at age 2.1?

DR. CLEMENS: Yeah.

DR. FUKAGAWA: That’s true.

DR. CLEMENS: And actually from a regulatory standard, is it geared for people two years of age, so it’s a regulatory thing.

DR. SLAVIN: I think that -- this is Joanne Slavin -- I think it’s a question, you know like if breast milk is 55 percent of calories come from fat --

DR. RIMM: It’s from fat.

DR. SLAVIN: So you start out on a high fat diet, and just, you know, for kids not getting our fat restrictions as tight as sometimes they are that kids really need to be on a high-fat diet, which kind of a conflict with obesity. You are like, well we’d better not do that, but --
DR. RIMM: Yeah. I mean, I think that the problem is there is not a lot of great data that kids who have more fat are more overweight. I think the problem is if they are put on pretzels, which have no fat versus how they process carbohydrates, and that leads to potentially to overeating. So, it would be a good area to look at. I don’t think there is a ton of data, prospective data on fat in kids. I could be wrong.

DR. VAN HORN: Well, having just been involved with an NHLBI pediatric panel, I think probably the biggest contribution to the data within the last five years has been the strip study recognizing that, you know, taking children from birth and actually having them on a lower, total fat lower saturated fat diet has yielded no adverse conditions, and in fact improved lipid levels in these children, who are now seven. So, the prospective data are really quite impressive, and I think could and probably should be reviewed and included in this presentation or in this discussion.

DR. CLEMENS: I see you point, Linda. I think there is some emerging data on cholesterol. We know that breast milk is naturally high in cholesterol.
DR. VAN HORN: Right.

DR. CLEMENS: And what the impact is neurological development as well.

DR. WILLIAMS: Chris Williams. Actually, from that strip study now they are showing lower rates of obesity in the children, so.

DR. VAN HORN: Yeah, right, exactly, both, both obesity and lipids. Right.

DR. WILLIAMS: And that’s with lower fats, low saturated fats and a little lower total fat.

DR. FUKAGAWA: But we also want to know what they are doing cognitively later.

DR. WILLIAMS: They are doing well.

DR. FUKAGAWA: They are doing well? Okay.

DR. CLEMENS: Good point.

DR. VAN HORN: Yeah. No adverse events at all on anything so far.

DR. PEARSON: One other point for discussion is the individual fatty acids, which fall under all of the unsaturated ones. Obviously there has been a reasonable amount done on elaidic, as kind of your poster child for your trans, and on oleic, obviously is
your arch typical mono, but does anyone else have any comments on specific fatty acid issues that you want to address, other than as the group, you know, mono, poly?

DR. CLEMENS: I think in dietary guidelines we obviously have a mixed message, and I will address that, when it comes to food safety. One side we say we should be consuming more fish so we get omega-3s; on the other side we are scaring people because of the methyl mercury story, and as a result, many OB/GYNs have advised their patients to stop eating fish when they are pregnant and when they are lactating. It would be nice if we could encourage the appropriate amount of -- and the ADA has done a wonderful job of trying to promote the appropriate servings and the appropriate types of fish during this vital period of development.

DR. PEARSON: Yeah. I was going to get to fish consumption in a little bit; maybe I will just hold my comments because we are going to get off that.

DR. CLEMENS: By all means. I would suggest that.

DR. PEARSON: All right. So here is the Lumper’s view of dietary fats, and that is that there have been
1 also research literature done on specific fat and
2 sterol nutrients, but rather, on food and diet-based
3 issues, which really do deal with conglomerative fats,
4 so this is -- and so fish is on the top of that list,
5 and certainly I have been impressed with the
6 epidemiologic and I think some clinical trial data on
7 fish consumption relative to neural development.
8 Certainly our environmental health group had a study in
9 Saychelles, in which the hypothesis that the relatively
10 high mercury fish that was consumed by that population
11 would impede neural development, and in fact the
12 relationship between fish eating and neural development
13 was significant and direct rather than inverse. So,
14 Mim, I was right, fish is brain food, it appears. And
15 so the other question is food-based is that there are
16 some other things in fish of interest than just the
17 omega-3 fatty acids. There is some taurine data and a
18 variety of issues, as well as a good protein source,
19 but I think our group was interested in taking fish on
20 as one of the, again a food pattern discussion rather
21 than an individual macronutrient. Any other comments
22 on fish? I think we should come up with -- I think,
Roger, your point about people being confused, I think you are correct on that.

DR. CLEMENS: And I think they are confused as well the type of fish that may be consumed to provide these types of healthful benefits; whether these are cold water fish, warm water fish; whether they are farm-fed or whether they are wild. We might be able to help in that regard in clarification.

DR. VAN HORN: Can I just ask a question that I honestly don’t know the answer to this? Because of the interest in fish and the fact that we keep advocating it, and you know, I’m all for that, is it within this group’s pervue to talk about the need for safe fish farming and the fact that, you know, as time goes on, if we want to keep recommending that the population consume more fish realizing that we have a limit and recognizing the importance of farming, that the mercury issue is, you would think, would be something that could be addressed in terms of safety of fish farming? I don’t know. Is that something that this group can --

DR. WANSINK: Sure. Yeah. This is Brian Wansink. Yeah. That’s a very -- within the pervue and it’s a
1 good thing to do too.
2
3 DR. VAN HORN: Okay. Great.
4
5 DR. RIMM: Yeah. This is Eric Rimm. I think there actually has been a fair bit of interesting data.
6 If you look at some of Emily Okin’s recent data on child development in mothers eating fish, that the mothers that ate fish it was very important for the cognitive development of the child, and if they ate fish and had high levels of mercury in their hair that actually hindered the development somewhat. So I think we can give a clear message that fish, you know, have been analyzed to death now. We sort of know how much mercury is in which fish, and it does vary a bit, but it’s not a perfect measurement, but we still do know that swordfish has a lot more mercury than salmon. So I think that, you know, I think that we can make a stronger statement than they could five years ago. Now we should probably do it in coordination with the EPA and everybody else who is trying to say the same thing at the same time, so that there is a clear message and there is not confusion.
8 DR. VAN HORN: Right. Right.
DR. PEARSON: Another food-based issue are nuts, and this obviously goes to omega-3, vegetable-based omega-3 rather than marine-based omega-3 alpha linolenic acid and some of those micronutrients, but it might be some other issues as well relative to those. But there is really quite a literature from small randomized trials certainly with lipid end points, and so this may be some comment, as well as it relates to omega-3 non-marine sources. Any comments on nuts?

Yes?

DR. APPEL: How about the whole issue of mothers being worried about allergy in children?

DR. PEARSON: Certainly there is with particularly peanuts, but I guess that would relate to other nuts as well. I’m not a --

DR. CLEMENS: Yes. We would include food allergies.

DR. PEREZ-ESCAMILLA: This is Rafael. You know, several scientists I remember before the last dietary guidelines were issued were calling for creating a separate group for nuts and not having together with the protein group, with everything together. And I
1 wonder if this is a question that this committee should
2 take on again at this time.
3
4 DR. NELSON: Maybe we should have a carbohydrate,
5 nut and protein, or protein, fat -- one committee?
6
7 DR. VAN HORN: That’s an interesting thought. One
8 thing I would just add is that was actually the comment
9 that I made earlier. It would take -- I did this
10 calculation. I think it’s something like you need
11 eight cups of walnuts to be equivalent to one ounce of
12 fish, in order to get the same amount of biologic value
13 omega-3. So again, the calorie, you know, contrasts
14 are huge, and so, you know, we would need to keep that
15 in mind too. Larry?
16
17 DR. APPEL: Yeah. Correct me if I am wrong -- oh,
18 this is Larry Appel -- I don’t think we actually, in
19 the 2005, looked at the epidemiologic data between nut
20 consumption and/or work on CVD. So, recognizing what
21 you just said, we didn’t -- it took a boat load -- a
22 more modest amounts of nuts, from what I understand are
23 actually associated with a reduced risk of CHD in
24 almost every study. So, we have to decide whether we
25 want to do it on, you know, have that as a research
DR. VAN HORN: Right.

DR. PEARSON: And I guess it was my contention that although certainly there are calories and carbohydrates in nuts, but I thought this was largely in the fat vehicle realm, in terms of what the bioactivities --

DR. SLAVIN: This is Joanne Slavin. I think of nuts more of a whole food, like whole grains that --

DR. PEARSON: I agree.

DR. SLAVIN: You know, fat is important, but there is fiber in it and other micronutrients, so that’s why it is so protective in epidemiologic studies is the package not any particular part of it.

DR. VAN HORN: Right.

DR. PEARSON: So -- well, maybe our Chair and Co-Chair can assign the nuts.

DR. VAN HORN: You are what you eat, is that what you are saying, Tom?

DR. PEARSON: Olive and canola oil are perhaps a little bit more clearer in terms of major sources of monounsaturated fats and obviously there have been a
number of trials with these, and obviously we have been advocating fruits and vegetables. I think there certainly are some obviously relative to low fat or certainly high monounsaturated fats diets, as well as protein sources that don’t have a lot of saturated fat associated with them. Obviously these have been some food-based issues.

In terms of diet-based, many of the specialty diets I think there have been some opportunities to reduce a variety of nutrients, calories, carbohydrates, et cetera, but many of these also focused on fats as a whole food. The Mediterranean diet particularly with high omega-3 fatty acids, high monounsaturated fatty acids, as a characteristic. The high protein, low carbohydrate diets obviously having to do with also frequently high saturated fats in the setting of everything, and then of course, the low fat, low cholesterol diets, which have been looked at, particularly given their higher carbohydrates, had some, you know, increase in triglycerides and decrease in HDL, as a negative consequence. Anyone who would like to talk about some of these specialty diets? We
hadn’t really talked about these in the various places, so one of the -- in an effort to be comprehensive and since they are out there and frequently used at a population level, I did want to bring it in, because it is, to some extent, is a dietary fat issue. Eric?

DR. RIMM: Yeah. This is Eric Rimm. I think one issue, and we don’t have to put names on them, but they are -- I mean, in terms of, you know, who sells the book to sell the high protein diet or a low fat diet, but the fact since the last Dietary Guidelines, there are now a number of trials obviously, including the Women’s Health Initiative, where there is -- you know, I think that there what will be of great interest, if we want to have obesity an end point, because in the end, most of them don’t work that well, and it’s really just how well you can stay on the diet that triggers the how well you -- how much weight you lose. So, I think it would be a good thing to look at, and I think we should focus more on longer trials than those that have just gone for six months, because six months you actually do see great differences depending on what trial you use. And so, it’s only where people can
sustain a diet for over a year or two years where you actually see that there is not great differences. So, I think that’s a very important thing to consider, and a lot of that research has been done in the last five years.

DR. PEARSON: Has there been consideration where to put those in terms of the review? Is that a whole -- is that a whole food issue? Because I think they are out there. I think that Eric is right in terms of the longer trials. Some of them actually come up with some safety issues as well.

DR. RIMM: Yes, they do.

DR. PEARSON: And so, like you said, a six-month look is oftentimes not adequate to look at some of these other issues.

DR. NELSON: Mim Nelson. Well, I guess I’m still advocating that we add yet another subcommittee that looks at behavior, meal patterns, things like that, because I think that maybe it’s too hard to put them into one of the other subcommittees, because it’s really more around the whole quality of the diet and the way we eat and gets at, you know, the ease. So,
I’m still hoping that we can think about a subcommittee that is innovative that way and then it would fit in this. And my sense with most of these diets is what you said, Eric, but it’s really you can create a really wholesome, whole foods kind of diet; whether it’s low fat, high protein, you know, it’s more the quality of the foods that are in it and whether someone can eat it for a long time. So --

DR. VAN HORN: Well, and just the whole question you just raised really, and I don’t know how much data there are on this subject, but the question of perhaps individual preferences -- I know certainly some of the work that Barbara Roles and others have done related to -- you know if you are looking for eating as much as you want, you know, then obviously you are going to go to a high complex carbohydrate approach because you get volume; but if you are going for, you know, intense flavor or something like that, perhaps then a higher fat type of diet but smaller portions would be your cup of tea, so to speak.

DR. NELSON: Right. Exactly.

DR. VAN HORN: So, whether there are data that
would differentiate for people, you know, a pathway
that as long as the total energy intake is reduced,
your, you know, your approach to it could be
individualized, as long as you don’t exceed your
calorie needs. That could offer people some hope, as
far as being able to eat the foods they really like
without having to, you know, totally compromise.

DR. RIMM: Yeah. I think there is no one diet for
everybody. I think there are 10 different things that
can work, and it just has to be something that you are
compliant with and that you are not eating too many
calories.

DR. SLAVIN: And I think it goes back to the
nutrient adequacy subcommittee too, that if you are on
a low carbohydrate diet, you can’t get enough fiber,
you know? So, it does affect nutrients; that even if
you are losing weight, if long-term, it’s not a good
diet because the nutrients won’t go along with you.

DR. RIMM: Yeah.

DR. PEARSON: Joanne?

DR. FUKAGAWA: Yes. I like the concept of lumping
for the diets, because I think we spent the last decade
or so really becoming more and more reductionists and thinking in isolation of one particular nutrient, one particular group, one particular vitamin or, you know, source, et cetera. And I think what we’re all hearing, at least I’m hearing, is that it’s really that integration of getting back to the basics of energy as conserved; that really, you know, how much you take in and how much you expend is really what’s going to end up with the outcome of better health. And so I think that’s one of the challenges we have is whether or not we continue to sort of stay somewhat unlumped or we find a way to lump the columns.

DR. PEARSON: Well, yeah, this is -- we should --

DR. NELSON: Maybe it’s a lumper’s subcommittee?

DR. PEARSON: Well we should get back to Mim’s proposal there and that is to really consider what we are going to do about these, because I think, at least my looking at the 2005, they were really very much reductionists, and it may be something maybe we want --

DR. VAN HORN: You know it’s possible and just one idea and the group can certainly discuss this or think about it as time goes on, because obviously we have our
plate pretty full just looking at the eight subcommittees that we have so far, but in some ways it sounds like, from all the presentations we have heard so far, that this is a cross-cutting issue that really interacts with every single subcommittee that we have. So perhaps one of the things that the science review group could take on, as well as bringing in individual representatives from each of these subcommittees is exactly that. These Guidelines mean nothing if people can’t follow them. And so it seems to me that, you know, we really owe it to the Secretaries, who both gave us that mandate, to come up with something simple, but you know implementable; that we would look at the behavioral side of these things as well. Larry?

DR. APPEL: Yeah, just one comment. I spoke with Trish Britten yesterday about our subcommittee structure. It turns out we actually needed a subcommittee on selected food groups and that emerged about halfway through the process, and so maybe starting earlier is better.

DR. VAN HORN: Okay. Good.

DR. PEARSON: Okay. In our one minute between us
and lunch, I did have one other view of dietary fats, and that’s the mechanistic view, and this actually has already been raised by Larry about the end points, and just to say that in the literature one could use the variety surrogate end points to look at the fatty acid effects. And, in doing this by subcommittee, I have already got a reasonable amount of feedback, and so I think maybe I didn’t put enough question marks on there, because there seems to be some difference of opinion, even just the three of us about some of these. Part of this has to do with relative to what, and a lot of this is relative to monounsaturated fats, which is kind of the standard versus more carbohydrates as the source of the calories. But the point of this slide I think really is the extent to which we are going to be interested in some of these metabolic intermediary end points. There are a number of studies of endothelial dysfunction which have come up since 2005. Certainly there was a very large literature, which has been added too relative to the lipoproteins. There have been some additional thrombosis studies; been a lot of studies on information relative to the interest in a variety of
bio markers, c-reactive proteins, other kinds, et cetera, but we should remember on the bottom line is that this is all a high-density, high energy density food and all of them have obviously the higher energy density that obviously it needs to interface with our caloric balance folks. Eric or Roger, any comments on mechanism?

DR. CLEMENS: You know, I think it’s really great that we have, are going to take on these particular areas of mechanisms, in particular, the general consumer is very interested in this inflammatory process. We see a lot of products out there about boosting the immune system, and all of us knows -- each of us knows that boosting the immune system is not what you always want to do. It would be wonderful if we had a good position, at least a collection of data to demonstrate modulation of the inflammatory processor of the immune system is appropriated, and from a dietary perspective this is how it could be done.

DR. PEARSON: Larry?

DR. APPEL: Yeah. I don’t know. I think we have to be very careful about this point, getting back to
the surrogate outcomes issue. I mean, I even think for inflammation, you know, we are not quite sure, you know, how to interpret these things, and we can, you know, we can spend a lot of time sort of, you know, doing literature searches on mechanisms, but never -- and not really change our final decision. So, I think we -- I like mechanisms myself, but I’m concerned about getting bogged down.

DR. PEARSON: So perhaps use this as more of a confirmatory or --

DR. APPEL: Biological clause.

DR. PEARSON: -- not the main agenda, but --

DR. APPEL: A biological clause is recommended.

DR. NELSON: Yeah.

DR. APPEL: You know, these are possibilities.

DR. PEARSON: Exactly. Eric?

DR. RIMM: I’d like to second that motion. I think -- this is Eric Rimm -- I think there is a lot of data now on clinical events and I think that’s probably the most important thing that we should be looking for. You know, the issue of N-6, you know, decreasing HDL and increasing inflammation, and yet there is a
plethora of data on benefits of N-6 and cardiovascular disease. So, I think that, you know, there are a lot more question marks that we can put here than arrows essentially, so I think that this may be good to support what we want to say, but we should stick with good end points where we have them.

DR. PEARSON: Other comments?
(No audible response).

DR. PEARSON: If not, I think -- I want to thank my partners on the subcommittee, Eric and Roger, for -- we had met before about this and the associated discussion. I don’t know. I have about 12 areas of recommendations we can certainly sink our teeth into. And I want to thank the group for their addition to this facilitative discussion.

One of the areas that I think we’d like some input on is outside individuals that we may want to bring in for a consultation here, and so we are certainly open to suggestions. Dr. Van Horn?

DR. VAN HORN: Right. Thank you. I think each of the groups is probably looking for similar input, so I think we all ought to be open to those ideas.
I want to thank the committee -- Tom, you and your group -- but everyone this morning. I think it’s been absolutely a rich and full, and incredibly valuable discussion, so I want to thank all the panelists for the excellent work they have done. And now we will all adjourn for lunch and return at 1:30. So thank you.

(Whereupon, at 1:14 p.m., a lunch recess is taken).

DR. WANSINK: Last week, at the American Dietetic Association, I was there and this person came up to me and said, I think you are sending a terrible signal by having the Dietary Guidelines Advisory Committee meet on Halloween. Ahhh. She says, just think about, just think about it. And I did think about it, and what I thought is that this is the only holiday that has its only food pyramid. So thank you very much for taking your Halloween and the trick-or-treating with the kids to be here today. Linda?

DR. VAN HORN: Well, on that note, I think we’ll talk about Ethanol. Okay. We are good to go. We are going to talk about Ethanol, and Dr. Rimm is in charge.

DR. RIMM: Thank you very much. All right. What
I’d like to do for my talk is take a format -- I’m also from the Midwest, so like Joanne I’m going to just do it as I was told. We are going to take the format of reviewing what was done in 2005, questions asked and conclusions from the Technical Report, and then go to the 70-page key recommendations, and then talk about is there new evidence for 2010, and if there is are there are there new questions that we could ask?

So, in 2005, several questions were -- the Committee came up with several questions, and I’ll put together the evidence to answer these questions. The first was, among persons who consumed four or less drinks per day, what is the dose response between alcohol and health? A pretty global question, but I think what came out of it was what is now, I think, quite well accepted in the scientific community is that one to two drinks a day lowers total mortality; one to two drinks per day is associated with lower risk of coronary heart disease; one drink slightly increases breast cancer; and alcohol risks and benefits do not differ between middle age and elderly people, but there is little if any benefit for younger people. This is
actually verbatim from the -- these are the words and
the word-smithing that went on to fit into each one of
these categories. So, I think this is important
especially the last point, because I’d like to bring
this up again when we get to the questions asked.

The next question asked is what is the
relationship between consuming four or fewer drinks and
macro or micronutrient profile in overall diet quality.
I think we have touched on this several times today
about looking at the overall diet, and the question
that was posed here was, if someone does drink four or
deeper drinks, does it actually impact the rest of their
diet. So not biologically what happens, but if someone
drinks, do they have an insufficient micronutrient
intake, or do they change the composition of their
macronutrient intake? And, what they found based on
national data is that individuals who drink one to two
drinks per day, it is not associated with an inferior
diet quality. And this was done through several
different measures, but specifically for macro and
micronutrient intakes or profile, they found that
people who drink don’t have necessarily any worse off
1 diets. I don’t think that’s a proper terminology, but
2 you get the idea.
3
4 So going back to the technical report into the key
5 recommendations, this actually made it in I think as
6 almost one of the next to last chapters, chapter 9 on
7 alcoholic beverages, and so the recommendations there
8 were those who choose to drink alcoholic beverages
9 should do so sensibly and in moderation. And,
10 interestingly, if you go back to the history of the
11 Dietary Guidelines, back to the first one in 1980, this
12 is one of the few guidelines that has almost not
13 changed since 1980. Again, there has been a little bit
14 of word-smithing that has gone on. Now it says
15 sensibly. I don’t think it said sensibly back in 1980.
16 But, for the most part, the guideline has stayed the
17 same. The wording that has come after it has changed a
18 lot. In just terms of -- just because there has been much
19 better science in the last 25 years to study the health
20 effects of alcohol.
21
22 And then consumption is defined as one drink a
23 day; moderate consumption is defined as up to one drink
24 per day for women and up to two drinks per day for men.
1  And again, that’s meant to be per day and not
2  necessarily an average per week where someone could
3  have all their consumption on Friday and Saturday
4  night. I didn’t mean for that to be a joke, but thank
5  you very much for joining in. Alcoholic beverages
6  should not be consumed -- again this is verbatim --
7  should not be consumed by some individuals, including
8  those who cannot restrict their alcohol intake; women
9  of childbearing age, who may become pregnant; pregnant
10  and lactating women; children and adolescents; and
11  individuals taking medications that can interact with
12  alcohol. And again, this has changed somewhat over
13  time as there has been more evidence, but for the most
14  part, in general, this has covered similar populations.
15  And then of course, those also with specific medical
16  conditions. Alcoholic beverages should be avoided by
17  individuals engaging in activities that require
18  attention, skill or coordination, such as driving or
19  operating machinery, or potentially sitting on a
20  Dietary Guidelines Committee.
21  So the definition for moderate drinking is defined
22  as 12 ounces of regular beer; five ounces of wine; and
one-and-a-half ounces of 80-proof distilled spirits. And in the technical report they go through the discussion of how much is consumed in this country and how that has changed over time. What I find is interesting, if you can see this, is this actually was in the 70-page Dietary Guidelines, is that several different beverage types are listed, as well as -- if you look at the last two columns -- the average portion size -- so that’s 12 ounces for beer; five ounces for wine; three ounces for sweet dessert wine and one-and-a-half ounces for 80-proof distilled spirits. And the last column is the calories and the calories, if you can read the small print way down at the bottom, are based on release 17 of the USDA nutrient database or standard references. So I looked this up, they are now up to release 21, and release 21 actually -- because the alcohol content of beverages has increased in the country, beer is now over 150 for a regular beer; red wine and white wine are now up at like 120 to 130 calories per same five-ounce serving, only because the amount of alcohol has changed. Obviously, if you are measuring 80-proof distilled spirits, it’s still going
to be the same calories because it’s 80-proof distilled spirits. So the question is, what are people consuming, and in fact, we should at least make, take this into consideration when looking at total caloric intake, if we are talking about average consumption of alcohol.

So the research recommendations for 2005 – and Larry mentioned that you and your committee at the time didn’t spend as much time talking about research recommendations, this was mostly saved for the last meeting so, you know, this may not be as important. If they wanted to investigate the relationship between alcohol consumption and obesity, this obviously is quite important, and the evidence they had concluded at that time was that there was insufficient evidence. It didn’t look like alcohol in moderation was associated with obesity. They also had some more policy issues, which was to investigate the impact of adding calorie information to the labels of alcoholic beverages, including whether, for educational purposes it would be sufficient to include only calories. I know there has been a movement afoot to do that. Thus far I don’t
believe that calories are added -- are mandated to be added to the labels of alcoholic beverages.

Investigate the impact of banning alcohol advertising, when and where it might increase underage drinking, during college sporting events, for example. This obviously is a very important issue that universities struggle with across the country as the problem of excessive or binge drinking among college students, either of age or not of age. And also, to investigate the impact of a unified federal message on alcohol and health through increased collaboration across agencies or consolidation of authority under one federal agency. I am guessing this is way beyond the purview of the Dietary Guidelines, but it was an interesting thing that was brought up in 2005.

So, 2010 Guidelines, is there evidence; are there new questions to be asked? I think there is a bit more evidence on drinking patterns. This is a challenging one, because when people -- when you say drinking patterns, the first response is, are you talking about binge drinking? And that is one aspect of drinking patterns. I think another aspect of it is people who
drink every day versus many who drink many days per
week but don’t drink every day. The issue is of, you
know, transitions from certain drinking, types of
drinking patterns to actually excessive alcohol
consumption and alcohol abuse.

Just a few things that we have looked at and our
cohorts -- this is a cohort of 50,000 men looking at
alcohol and coronary heart disease -- and on the bottom
axis you can see the number of days they reported they
consumed alcohol, and you can see that the most benefit
came when you drank at least every other day, and there
wasn’t additional benefit from drinking more days. And
even among the categories of days per week, you can see
the amount they were drinking actually didn’t make that
much difference. It was mostly important to get
alcohol in the system at least every other day to -- I
shouldn’t be saying that with a toxicologist here --
it’s not meant as a pharmaceutical, but more as people,
you know, who were drinking at least every other day
were getting a benefit, and drinking beyond that there
was not further benefit. We also looked among people
that were the healthiest of healthiest in our cohort.
Of the 50,000 men, only 3,195 men were not overweight, didn’t smoke, had a healthy diet and had regular exercise. Even among those men there was about 200 cases of MI (myocardial infarction) over the course of 16 years of follow-up, and we still found that days per week of alcohol consumption lowered the risk of heart disease among these very healthy individuals, who otherwise didn’t have standard risk factors.

So, I put this slide here mostly to remind me of the other question, which is, is there new evidence of health effects in vulnerable populations? That was among the healthiest of healthiest, but with 60 percent of the population overweight and more people with diabetes and hypertension, there hasn’t been, I don’t think, necessarily a single message of what to do, what to tell people about alcohol among individuals who are otherwise compromised or at higher risk for other chronic diseases.

This was a recent metabolic study or a clinical trial of alcohol that was done in Israel, and it’s interesting. They actually randomized people who seldomly drank alcohol, so they weren’t lifetime
abstainers, but they were people who seldomly drank alcohol, and they were randomized into either one drink a day on the right side, or a controlled beverage which did not have Ethanol in it. And this was just looking at their fasting plasma glucose. And you can see that on the left side in the control group, they started on average at a fasting glucose of 136.7 and went to 138.6. These are among all diabetics in Israel, so as it is, their glucose levels are quite high. The control group was not affected by alcohol. If you look at the right side, those that consumed one drink per day, they had about a 20 milligram per deciliter drop in their glucose after a 12-week period. So this is just to say that it is interesting to think about the impacts of alcohol among a high-risk population. The American Diabetes Association actually takes on the same stance as the Dietary Guidelines; that is, those who drink can do so in moderation.

Health effects of new drinkers. This is a very challenging question which we may not ever have evidence from a clinical trial looking at the impact of alcohol long-term among new drinkers. There was a
recent study published from the ARIC study. This is not my study even though the name is quite similar. It actually stands for Atherosclerosis Risk In Community Study. It’s done from four different communities around the United States representing different ethnicities. And it’s a little tricky, but essentially what they had is a lot of -- they looked at the non-drinkers at baseline at visit one, and visit three six years later, actually some of them had started to drink, either moderately or heavy, and they were able to follow them after that time period to see who developed coronary heart disease. And, believe it or not, there were some people who were middle-aged, who started drinking who previously had been non-drinkers, and if you look across at their odds ratio among moderate drinkers, their odds of developing heart disease was .62. So this was, I think, one of the first studies that had enough statistical power to look at what happens if non-drinkers take up moderate drinking in mid-life. I had very few people who became heavy drinkers, and therefore the comparison levels were quite wide, .41 to 4.9. So really I can’t think
-- I think we can’t draw much from this. The author’s conclusion from this was that there aren’t many people who become heavy drinkers, who convert -- who start drinking in middle age.

So, you know, there is not enough data here to come up with a strong statement, and I don’t think we ever would come up with a statement that people should start drinking alcohol, but at least there is some evidence that among middle-aged individuals there is not a lot of people who go on to become heavy drinkers, who started moderate.

So, new questions to be asked? We had a quick circulation of emails among individuals on the committee, Larry Appel and Tom Pearson, and I guess some of the challenges, one of them that I thought we should look at is that, well one to two drinks per day is not associated with a poor quality diet, as concluded in the 2005 Guidelines. Biologically alcohol does impact a lot of important metabolic systems, and specifically we know, even though you may have the same diet, that drinkers actually may not absorb folate as well or alcohol may actually interfere with use of
folate. And there is a number of other biological systems, which I think there is now beginning to be enough data that we could look at. I don’t think we would make special, necessary special dietary guidelines for drinkers, but I think it’s enough to be -- there is enough data out there that we should be at least aware of the fact that alcohol does, even at moderate levels, interfere with metabolism and absorption of micronutrients. So that may be one area that we can ask.

The second, another area that we thought of is that, who should not drink. I provided a list of groups or categories of individuals that should not drink, and there may be growing literature on other individuals or people with certain family histories that also should be given guidance potentially not to drink or to drink less. And I think related to that, are the Guidelines explicit enough on contraindications for alcohol consumption?

The next area which I guess maybe I disagreed with the Guidelines in 2005 is, does one to two drinks per day really have little if any benefit for those less
than 30 -- less than 45 years of age? That statement was based mostly on the benefits for cardiovascular disease, and there is very little cardiovascular disease among individuals less than 45, so while I can see where that conclusion came from, if we think about most people who have heart disease when they are 50 or 60 or 70, if they are getting benefit from alcohol, it’s very unlikely that all of them started drinking when they were 45. It’s more likely that they started drinking at an earlier age where they already were accruing some of the benefits; the increased HDL cholesterol; the decrease in clotting; and the probable benefit on atherosclerosis. So, I think that there probably is -- cardiovascular disease takes 30 or 40 years to develop, so I think to say that there is only benefit among people over 45 may be too broad of a conclusion.

But also importantly and related to the headlines today on diabetes, there is now at least 10 studies, which show prospectively that individuals who drink one to two drinks per day have about a 30 to 40 percent reduction in risk of developing Type 2 diabetes. And
that may be related to lower levels of glucose, an
increased insulin sensitivity related to moderate
alcohol consumption and alcohol blunting the glycemic
effect of a meal.

So, that’s sort of where we stopped. There
probably are other questions that could come up with
alcohol. I don’t expect that the bottom line will
change much but I think we could explore some
questions and potentially expand our guidance for some
certain subpopulations and for certain age ranges. So,
I will leave it there. I hope I went quick enough to
stay within my allotted half hour Ethanol time?

DR. VAN HORN: Excellent. Does anyone else on the
subcommittee have anything, Larry or Tom?

DR. APPEL: Yeah. This is Larry Appel. I --
thanks, Eric. I had one question that’s a little bit
out of the box, but it pertains to the fact that
alcohol is something that you are not supposed to
consume and then you do consume it, and we know that
this transition period is a big problem. And one
question that, if I could, I threw out, to paraphrase
this, are there healthy patterns of starting drinking
and what are those, which is a little bit different, but it relates to behavior issues, but that might be something we should at least put on our list to consider.

DR. VAN HORN: Good point. Tom?

DR. PEARSON: For the nutrition committee of the American Heart Association, I wrote some guidelines about 10 years ago on this subject, and we actually did an analysis addressing the CDC’s recurrent information about 100,000 excess deaths in the United States due to alcohol consumption every year. Looking at also if we assume that everyone were a tee-totaler, what would be the effect, and it’s about an 86,000 excess deaths. So it’s about a wash. So you have this U or J, or whatever you want to call it, on both sides of it. So it makes it, for a messaging issue, very difficult.

The problem is that the MI benefits are all in the middle-aged and older individuals, and many -- not all -- but many of the auto accidents, violence and issues are in the younger people. So this issue that Larry raised I think is very important, and I may need to go up one more step and maybe at this point look at the
evidence to say, are there any interventions in which young people’s behavior can be changed so that they are using this as a beverage and not a drug? Because that’s really the issue. Is it the -- the excess mortalities is when alcohol is used as a drug and not as a beverage. And I think what the nutrition guidelines ought to do is to see as many people who could be curtailed from using this as a drug and moved over into the one or two drinks a day and really have it all beneficial. So, I would go one more step than what Larry said, and that was not only to the observational studies, but if there had been any interventions.

DR. VAN HORN: Very good.

DR. ACHTERBERG. Cheryl Achterberg. I liked those comments. And this might be a marginal addition, but nonetheless if we are going to be looking at these drinking patterns and so forth, it might be worth noting somewhere that there are over 21 university presidents now working together to create a recommendation to lower the drinking age to 18. So as this committee is working, we might want to be
monitoring that, if we decide to make a statement.

DR. WILLIAMS: Christine Williams. I have a question about, with the growing numbers of overweight and obese individuals and the non-alcoholics data on hepatitis, what is the effect of moderate alcohol intake on those individuals?

DR. RIMM: Yeah. You know, I think that’s a really important question. I think there is sort of two separate issues biologically what’s going on, I mean, what has been studied in very clinical detailed clinical studies, and then epidemiologically, what do you see if you study hundreds of thousands of people? And the hundreds of thousands of people ultimately are at risk for diabetes first, and in those populations alcohol actually is beneficial when consumed in moderation; and when not consumed in moderation actually is detriment. And so it is still -- that has not changed. The underlying population at risk has changed, and so generally what you see is a greater benefit than you would have seen in a lean population, but you also see potentially greater risk, because they are already at -- they already have (inaudible) related to
obesity, so it makes it even more of a challenge to
come up with sort of a simple, you know, phrase that
you can do to capture the entire population.

DR. VAN HORN: Tom?

DR. PEARSON: Another implementation issue really
is to not only look again at the algorithms or
whatever that would identify people who shouldn’t drink
at all. I mean, certainly there are some people who
shouldn’t drink at all, but the extent to which they
are actually implemented, I mean I think -- I think
most people find out they shouldn’t drink at all by
drinking excessively and then ending up in our
hospitals with pancreatitis or addicted to alcohol, et
cetera. And it would be helpful if we could come up
with ways that work so, you know, we don’t have that.

DR. FUKAGAWA: Is it also -- this is Naomi -- is
it also possible to consider where these three carbon
fragments go in terms of its impact on lipogenesis or
lipid metabolism, because you know it is another source
of Acetyl CoA, which could go down fatty acids side? I
don’t know.

DR. PEARSON: I think the issue is lipogenesis
that we need -- it affects lipogenesis about 14
different metabolic steps and --

DR. WILLIAMS: Yeah. Okay.

DR. PEARSON: I think that’s pretty well worked out. What I am not sure is that unless there is some new health effect of one of these carbon fragment metabolites that I don’t know about -- I think it’s been pretty well identified. You are right it is a source of Acetyl CoA for sure.

DR. WILLIAMS: So having it as a source -- okay.

Yeah. Yeah.

DR. PEARSON: And obviously there is a lot of empty calories there, but I’m not sure there is anything new.

DR. WILLIAMS: Right.

DR. RIMM: Right. I mean I think that’s what the last Dietary Guidelines started with is, you know, are there empty calories, does it really impact everything else?

DR. WILLIAMS: Yeah.

DR. RIMM: They are not going to be worried about the fact that people are displacing it for fat,
carbohydrate or protein. It didn’t look like that when you are drinking in moderation. Obviously that’s not the case when you are drinking more, and it does start to impact metabolism and fatty acid metabolism, as well as displacing micronutrients and interfering with micronutrient absorption. So, I think at the high end, the message definitely shouldn’t change. The message was quite strong in the last Dietary Guidelines about drinking excessively, so.

DR. VAN HORN: Good. Any other comments? All right. Very good. Thank you very much.

And for our last discussion this afternoon, we are going to hear about food safety and technology, and that will be Roger and Rafael. Right. Okay.

DR. CLEMENS: Thank you, Linda. This is a joint project with Rafael, so we have a good space of population representation, with any luck at all. We realize too that food safety is paramount and everyone wants to be responsible and everyone should be responsible. We noticed that in the opening remarks by both Secretaries and the Under Secretaries that food safety was part of their presentation. It’s on the
tips of everybody’s tongue. It’s a major responsibility within all the major agencies, so we -- from farm to fork, from good agriculture practices to the dining room table, food safety continuum is everyone’s responsibility.

As we look at the Dietary Guidelines that we presented last 2005 certainly these are the major bullets that were presented in that fine report. In this particular case we look at separate, as Rafael and I collaboratively discussed the options, we find that particularly in low income areas that the separation and the appropriate use of utensils and cutting boards alike sometimes don’t permit or do not execute the separation of foods, therefore leading to contamination between raw foods and contamination of cooked foods, and that’s totally inappropriate.

The Guidelines do a very nice job in outlining what the responsibilities are and what each step represents. From an international perspective, these are adopted icons. We notice that the last report did not use these icons. They were just recently developed. As I and Rafael have traveled
internationally, we find that these icons are now being used more and more in various professional organizations and in restaurants and hotels alike to encourage the locals to wash their hands; to wash food contact surfaces and so forth. Importantly we find that a temperature and time of proper cooking is an issue, both domestically and internationally, and certainly this is a big message that has been purveyed across the consumer groups. What we see now is we look at a number of groups and the last icon is showing the temperature range which is considered safe versus the temperature range which is considered dangerous. There has been a change in that range in the last five years, which would be pertinent information to be discussed and presented for the next Dietary Guidelines.

Another wonderful project that was supported by the last Dietary Guidelines and also with the FDA, the USDA is -- these various bullet points -- clearly the food storage and spoilage we often forget about spoilage organisms, and if you look at the history of food usage that many cultures, and depending on your lineage and your heritage, you will see that you may
have a spoiled cheese, but often we fail to discard it. We just cut off the obvious and get rid of it and don’t get rid of it rather. We know that can cause some issues as well. We are very pleased to see that listeriosis was brought to the attention the last time, and we want to look at other microbes that may be affecting the food supply as well, and particularly in the home. As we mentioned earlier, that methyl mercury and one area that we examined most closely and we hope that we will examine more carefully this time around as well is that one side we are saying we are concerned about the methyl mercury in fish and the other side we are saying we should be consuming more fish for a number of health reasons. In my own case, and my daughter just delivered our first grandchild, her OB/G said don’t consume any fish; at any other time we say we should consuming fish. So you see we get mixed messages on the healthful benefits of fish because of the potential methyl mercury.

There have been a number of new data that have come out that are now available and it’s incumbent upon us to re-examine those data to health risk benefits.
And a really nice survey that was conducted recently by the International Food Information Council published just earlier this year, you will see that some of the messages in terms of food safety preparation in the home, and in fact that message is getting out to the audience and we are really pleased about that. We see that people are now washing their hands at least more frequently. And when it comes down to one of our pets, that is, Rafael’s and mine, that separation is not quite clear amongst the population and group. And this we suspect is quite obvious regardless of your socioeconomic status in various cultures. We need to identify this in some way to communicate this more effectively to the population groups.

Very few are using the thermometer, whether they have it over a grill or in a microwave. You see it down there at the bottom of this particular graphic. Thermometers and microwaves historically did not mix, yet today’s technology says that thermometers are quite available, and in fact, you can monitor the internal temperatures of foods that are being cooked and cooked.
Overall, what are the consumer expectations in terms of food safety? The consumer expectation is that all food should be absolutely safe. A wonderful ideal -- not possible. We know that all foods carry a natural or unnatural -- carries some form of risk, therefore, the other button that is pushing loudly with most consumers is it’s all natural. Most consumers feel today that natural means safe. Those of us involved with food toxicology are realizing the fact that natural does not necessarily mean safe and we can identify a number of those areas. This may be an area for us to add to the chapter in terms of food safety.

Consumers want convenience and yet fresh. Sometimes those are like oxymorons. What is fresh? It has not been defined. But they do want convenience, and partly -- and maybe part of that, they want to cook a meal in ten seconds and then enjoy that meal. And that’s probably with fast food, rapid preparation, hurry up and go. We need to sit down and really enjoy the meals together like we have enjoyed the lunches together.
Positive messages? There are a lot of negative messages relative to food or food components. We would like to consider a positive message what foods deliver, and today this group is looking at foods in total as a positive way to deliver food messages.

We know that there are a number of technologies to improve the quality of life, as well as the quality of food. We are going to address that in the next graphic, but also address the movement, in terms of locally grown, in an effort to control costs and food quality. Well some of those issues are right here, in terms of the home kitchen is the last line of defense. It’s up to the individual -- it could be -- well, the home kitchen, it could be the back seat of the car that also has some impact on food safety.

Well a number of big organizations are conveying a message, and our hope is that we would have a message in food safety that is consistent with this and supported by the National Restaurant Association, in terms of serve safe all restaurants, all professional chefs, all major food companies that are involved with this safe serve application. And then various messages
here we want to be sure we get our data out to the
general consumer as well.

In addition, there is a great website, wonderful
information available through www.foodsafety.gov,
wonderful pull-down messages. Again, we want to be
certain that we have continuity and harmonization
messages to be delivered between the report that we are
generating and the messages that are available at this
website.

Well, where are we going? The technology will
hopefully help the consumers of tomorrow and the
consumers of today with these type of tools. We know
that the films and the saran wraps, if you will, of
today will be different of tomorrow. We know that the
packaging materials will be different tomorrow. Those
packaging materials and wraps and films will actually
improve stability. They will include oxygen
scavengers; they will include various sensor noses, if
you will, so they will automatically tell you whether a
product has spoiled. It will tell us the heat shock,
the heat exposure and thermal profiles that our foods
have undergone so that in fact we can ensure a safe
food supply, particularly in the home. At the end of the day we want to be sure that food safety drives the technology and resources, sanitation resources that are in the kitchen.

And lastly, we want to know, perhaps not addressed by the last group, that is, what is natural and does natural always mean safe? Can we give some guidance in this area relative to the safety of natural foods and really educate the consumer about this very important topic. We know very much that organic foods have received about four percent or so penetrations in the U.S. market. The national organic program is very helpful in educating the consumer, as well as the farmer, and yet we also know the research that I have done and many other -- and Joanne brought up some of this information yesterday -- that organic does not necessarily mean safe. And the only work that we have done at the USC, examined the bio burden of organic and in conventionally farmed foods, and in this particular case we demonstrated in many cases that organic foods have a much larger bio burden than those which are conventionally produced.
We want to look at that. In addition to some of the methodologies that are being used today to assess microbial loads, we note that we found at least that many of the organisms that we see are much slower growing than the BAM actually will detect, and could that in fact suggest we may want to look at additional methodology, maybe outside the pervue of this group, but yet it is something for us to discuss.

Buy local -- this is clearly a big movement across the country and around the world. While we support the buy local movement the buy safety is really more important. We know that many of the farms do not practice good agricultural practices. When you ask them in the farmer’s market, what is organic, they don’t know what organic means. They just display the placquard. So we need to educate the farmers, as well as educate the consumers, when they buy local be sure that they buy safe.

Another area that’s important to us, and this is mentioned by the opening remarks by the Secretary is that the generation, the baby boom generation is about to retire. In that retirement generation we see that
these individuals are polypharmate; they are taking multiple medications. The message of the food interaction, particularly the nutrient interaction with the medications is not well-known. I reflect back on the days that Daphne Rowe started wonderful work at Cornell. Unfortunately, a traffic accident took her life much too soon. We know that antibiotics certainly deplete the microfluora in the G.I. tract. We know certain that antihypertensive medications obviously affect electrolyte balance. We know that some antibiotics affect the mineral absorption. We know that some anti-seizure medications affect folic acid metabolism. The length of and degree of what we know about medications is growing. Maybe we have an opportunity here to educate the aging population so that as they take medication, we can work with the professionals and provide this great educational opportunity.

And lastly, well who can do it? Well, we know that Dr. Carl Winter, who is a noted food safety expert and toxicologist has a good handle in really studying the dynamics of the increased fruits and vegetable and
grain production and consumption may have in terms of exposure to not only environmental toxins, but in toxins that are naturally occurring in foods that you and I have come to enjoy.

In addition, Dr. Peter Preston, who is a Lieutenant Commander with the U.S. Navy, with the Medical Corps in Jacksonville, Florida, has considerable expertise of food drug interactions. It’s those kinds of interactions that we need to have a better grasp.

With that, Rafael, any remarks, please?

DR. PEREZ-ESCAMILLA: Thank you for an excellent presentation, Roger. I want to add that although for some food safety behaviors the awareness may have increased in the continuum of consumers, national surveys continue to show that we consumers underestimate considerably the risks of potential food-borne outbreaks at home. And what complicates matters very much is that a lot of these outbreaks are never reported, so from the epidemiological point of view, we need better methods to do better monitoring and surveillance of these food-borne illness outbreaks at
And this is very important, because if this data confirms that a very good percentage of food-borne illnesses originate at home, that is likely to change the attitude of consumers towards to food safety, and our research shows with microbiological health outcomes that the attitude that people have towards food safety matters in terms of their practices. So, in terms of changing food safety behaviors, addressing changes in the attitude towards the risk of home-based food-borne outbreaks I think is -- food-borne illness outbreaks is important.

I find the messages and recommendations from the 2005 Dietary Guidelines that I have in front of me very complex. I think we need to do a better job at explaining to the public what cross-contamination means. If you saw in the data presented by Roger that separation which is aggressive cross-contamination is one of the behaviors that is practiced the least and probably not understood very well. Thank you.

DR. CLEMENS: Thank you, Rafael. Comments?

DR. VAN HORN: Tom?

DR. PEARSON: Yes. Is there an issue of the
globalization of the food supply -- this is Tom Pearson
-- the globalization of the food supply and issues that
you are going to bring up relative to food safety? I
mean, obviously a number of both environmental and
individual product safety issues might be attended to
here, but not necessarily elsewhere, so.

DR. NELSON: Could I? I mean, I would just add, I
don’t know that it’s the globalization, but it’s also
the sort of the food processing, the way, I mean
especially meat and other things, you know there is a
real food safety issue that -- it’s sort of -- I mean,
it’s sort of lumped together with that. This is Mim
Nelson.

DR. CLEMENS: Sure. The same issue. Yeah.

DR. NELSON: Yeah, the same issue. Just, I am not
sure we want to link organic and local food in the food
safety piece. I think it’s another issue. I think
overall food safety cuts across all types of foods.
So, I think it might be a little bit sort of strange to
put them in there. I don’t know.

DR. VAN HORN: Go ahead.

DR. POST: And I thought you were getting at issues
like nitrates, you know? Is that something that -- I mean, I don’t think that’s covered any other place, you know. Does, you know, barbequing certain meats lead to a certain, you know, an increased risk of certain forms of cancer.

DR. NELSON: I think that -- this is Mim Nelson again -- but we spoke a little bit in one of our subcommittee meetings about phone calls just, just all the things that are being added to foods in terms of the functional foods and everything else. I mean, a lot of them we don’t know what the safety is and that seems like it probably should be in this committee. Would -- I mean not that we are trying to give you more work, but it’s, you know, it’s the food additives of all sorts I think is the range.

DR. CLEMENS: Yeah, and maybe -- this is Rog -- maybe the food additives -- this is really interesting, all of the food additives we see here in the United States are of course considered safe. What’s interesting now is that more and more we see a number of herbs and spices and the blends of some of these things are almost at the pharmacological dose. And as
we look at the pharmacology of some of the components and the mixture of these components that may raise a number of eyebrows and deserves further attention.

DR. FUKAGAWA: This is Naomi. I think I think back on Julia Child’s later interviews and she was asked, what has changed the most about eating and food in her career, and she said it was really the issue that people have become afraid of their food. And that’s perhaps the biggest change that she has seen, and it’s more than just the food safety, but being afraid of fat; being afraid of salt; being afraid of, you know, insufficient this or insufficient that. So I think if we do something that’s potentially valuable for the public, it would be trying to narrow and simplify our messages.

But I would like to follow-up on Mim’s thing about organic and lumping together local food and, you know, your concern about bio burden, and that in many ways some of the issues that we are dealing with is the fact that the boomers have gotten even afraid of microbes. And microbes and bio burden is not necessarily bad for general health, and in some ways, some of the problems
we have encountered, especially with resistant
bacteria, you know, in the hospitals and things are
related to over concern about this type of hygiene. So
I think we have to sort of try to balance, you know,
that entire message.

DR. PEREZ-ESCAMILLA: This is Rafael. I want to
also mention that as we are aware there have been
recent of food-borne outbreaks related to the
consumption of fresh vegetables, and some of that food
has come from outside. Some of those outbreaks, like
the spinach one, was food grown in the U.S. But the
point is that the washing, proper washing of the fresh
produce I think is an issue that is much more urgent to
be addressed now than it was perhaps even five years
ago.

DR. SLAVIN: This is Joanne Slavin. I think
especially as we promote fresh, we forget about that
food processing cleans things out, you know, like why
-- people always say, why did we take whole grains and
make them less healthy? Well, whole grains from the
healthy are not very clean, so there is a lot of
processing that’s really positive, so to not lose sight
DR. NELSON: Mim Nelson. I have a question. Where are we going to be dealing with all of these things that are now added to a lot of different foods, like isolated isoflava -- I mean, isoflavones -- and soy protein? I mean, there is like all these things that, because there has been one study or something that are now added, and I think it’s a real concern of mine, because we don’t really know some of the safety of these -- especially for different populations. Is that going to be -- Sorry, I keep adding stuff, but is that in the food safety, or would it be in the nutritional adequacy? It seems like it’s a food safety question, but.

DR. CLEMENS: It could very well be, because we don’t have an RDA or DRI for these things --

DR. NELSON: Right.

DR. CLEMENS: -- so they will fall into these functional food components, if you will, the bioactives that everyone has spoken about, so maybe food safety is to examine some of the bioactives that are really for which most of the consumers are concerned. For
example, we know that the American Heart Association pulled back their support for soy earlier this year, if I recall correctly, in March, if I remember right. So, they said, do we change some of that? Do we want to educate the consumer on behalf of the latest information? Maybe this is an opportunity for us to examine it. Good point.

DR. NELSON: Right.

DR. VAN HORN: Yeah. I think that’s an excellent idea and to move it ahead. Robert?

DR. POST: If I could suggest too, to consider food safety then has a couple of perspectives and perhaps it’s evolved to be more than just microbes, and you are talking about food components that might very well have adverse health affects and that will cause sensitivities, and that includes allergens.

DR. CLEMENS: Yes.

DR. POST: So that might do it. Those might be areas to consider, and even broadening the view of what food safety involves.

DR. NELSON: And genetically-engineered foods now that we have the potential of purple tomatoes.
DR. POST: Well, internationally food allergens is a very big issue and now there are icons, it may be another way to educate consumers after we had a regulation in 19 -- 2006, if I remember right. So if we include allergens in cross-contamination again with that particular area is going to be important.

DR. VAN HORN: All right. Well, I want to thank the committee. And we have now come to the point where we need to express our consensus about whether we believe that this Dietary Guideline review should continue, and if there is enough evidence that we think we need to pursue a new set of guidelines. Is there a proposal, I guess, a motion from the group? Is that what we want?

DR. CLEMENS: We move to we need a new set of guidelines.

DR. VAN HORN: A new set of guidelines. Is there a second?

DR. PEARSON: Second.

DR. VAN HORN: Okay. Any discussion? Does anyone have any last minute comments on anything that we have said today?
(No audible response).

DR. VAN HORN: Okay. Well, with that I guess we’ll just do it the old-fashioned way. All those in favor, just raise your hand.

DR. VAN HORN: All right. Very good. It looks like it’s unanimous, we believe we should go forward with a new set of guidelines, and we will do our best to be up to the challenge. I’m sure I speak for everyone here. I guess our next meeting will be sometime in January. We have just been talking about that, and while the official dates have not yet been expressed, that will be coming up. It will be toward the end of January, we think about the 28 or 29, but those plans are still underway. And I guess with that I just want to thank everyone and --

DR. PEARSON: Subcommittees.

DR. VAN HORN: -- subcommittee discussion we will present at this point, or just who the subcommittees are. I guess the audience is not aware of who they are. So, pretty much it has to do with the same group that was presenting during these last two days; the Chair of the Energy Balance and Weight Management Group
is Dr. Pi-Sunyer, with Drs. Nelson, Williams, Perez-Escamilla, Slavin and Achterberg on that one; Food Safety and Technology is Dr. Clemens and Dr. Perez-Escamilla; Fluid and Electrolytes, the Chair is Dr. Dr. Apple, with Drs. Williams, Pearson and Nichols-Richardson.

DR. NICHOLS-RICHARDSON: Can I make a request?

DR. VAN HORN: Sure. To come off of fluid and electrolytes since there will be quite a bit in nutrient adequacy, if that’s -- if there is no objection?


DR. NICHOLS-RICHARDSON: Okay. Thanks.

DR. VAN HORN: We will remove you from that one. Ethanol is Dr. Rimm with Drs. Appel and Pearson; Nutrient Adequacy, the Chair is Dr. Nichols-Richardson with Drs. Fukagawa, Achterberg, Slavin and Nelson; Fatty Acids, the Chair is Dr. Pearson, with Drs. Rimm and Clemens on that; Carbohydrates, the Chair is Dr. Slavin with contributors, Drs. Achterberg, Pi-Sunyer, and I’ll help out a little on that one; and then our
Science Review Committee will be myself with Drs. Fukagawa, Appel and Pi-Sunyer. And that represents the subcommittees that we will be convening in between now and the next meeting, and we look forward to launching in those directions and --

DR. NELSON: Can I comment?

DR. VAN HORN: Yes.

DR. NELSON: I still want to put a plug in for consideration of adding a subcommittee on behaviors and food patterns. And we might consider -- we might just wait until the next meeting to discuss that. We have time, but.

DR. VAN HORN: Right. I think that’s a good point; however, I also feel that it is the charge of every one of these subcommittees to take on a behavioral component, because it seems to me, and I think we discussed this earlier, that each one of them has some aspect of translational effort related to it, and therefore, it should be addressed in each of the subcommittees as well. Anything else?

(No audible response).

DR. VAN HORN: All right. Well, I guess we are
adjourned. Thank you very much. Have safe travel.

(Whereupon, at 2:23 p.m. the hearing concluded).
CERTIFICATE OF COURT REPORTER

I, NATALIA KORNILOVA, the officer before whom the foregoing was taken, do hereby certify that the following was taken by me by audio recording and thereafter reduced to typewriting under my direction; that said transcript is a true record of the recording taken by me; that I am neither counsel for, related to, nor employed by any of the parties to the action in which this deposition was taken; and, further, that I am not a relative or employee of any counsel or attorney employed by the parties hereto, nor financially or otherwise interested in the outcome of this action.

NATALIA KORNILOVA